# CLINICAL DIAGNOSIS

## CASE EXAMINATION AND THE ANALYSIS OF SYMPTOMS

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*"Felix qui potuit rerum cognoscere causas."*

# *PART III.*

# SYMPTOMS.

*In the practice of medicine the PROBLEM OF DIAGNOSIS is often put before the physician in the following manner: A patient comes to consult him because of some concrete abnormal condition, he is coughing, he is losing weight, he is sleepless, he has spat up blood, his skin is yellow, he has "kidney" pains, his skin itches, he has attacks of fever, he suffers from headache, he feels tired, etc. This presenting symptom must be traced back to its underlying cause. This is done by means of a verbal and physical examination which enables the practitioner to group about the principal symptom—principal at least in the patient's estimation—the remaining necessary data, signs, and symptoms;—in short, by a mental correlation of these data.*

*In the succeeding presentation of the subject a plan closely following the observations of ordinary practice has been adopted. In it have been collected the most frequently encountered symptoms, and in relation to each of these symptoms, after a brief review of the related anatomical and physiological features, the author has endeavored to explain how, with the assistance of the previous or concomitant accessory symptoms, and in the light of these data, a concrete diagnosis may be arrived at*

*Wherever it has seemed possible, each section has been summarized in the form of a diagrammatic, mnemotechnic table.*

# ALBUMINURIA.

[Albumin, *white of egg*—οὐρεῖν, *to urinate. Presence of albumin in the urine.*]

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Albuminuria is a sign which should never be allowed to pass unnoticed. The test for albumin in the urine should be carried out as regularly and routinely as auscultation of the chest or palpation of the abdomen. Two observations will give an idea as to its frequency of occurrence Out of 1000 subjects of both sexes and of all ages, the great majority suffering from chronic conditions, examined in the author's office, 204, *i e*, about one-fifth of the cases, showed albumin. In over half of these persons albuminuria had not previously been known to be present. Out of 1000 soldiers from twenty to forty-eight years of age under observation in a hospital, the great majority suffering from acute or subacute conditions, the author found 128 instances of transient or permanent albuminuria, constituting about one-eighth of the entire number.

Thus, albuminuria occurs with extraordinary frequency and under the most varied clinical conditions. Its symptomatic value, while sometimes practically *nil*, may be very great. In finding our way through the etiologic and pathogenetic maze of the various forms of albuminuria, the didactic presentation of the subject by Castaigne in his work entitled "Livre du médecin" (section on diseases of the kidneys) will be extensively availed of.

Clinically, *albuminuria* occurs in the form of:

I. *Acute albuminuria,* always symptomatic of an acute or subacute, infectious or toxic, nephritis which is comparatively easily diagnosed.

II. *Chronic albuminuria,* which, on the contrary, as we shall presently see, arises from a great variety of causes and the etiologic diagnosis of which is often a matter of considerable difficulty.

## ACUTE FORMS OF ALBUMINURIA.

**Acute albuminuria** is met with almost exclusively in the following four groups of cases. (*a*) Superficial and transitory acute nephritis; (*b*) typical acute nephritis; (*c*) hyperacute nephritis; (*d*) acute exacerbation in the course of a chronic nephritis.

(*a*) **Superficial and transitory acute nephritis** is a clinical type which is mild and very common and the presence of which should be ascertained through systematic examination of the urine in all *infectious* or *toxic diseases* (sore throat, grippe, pneumonia, acute gastric indigestion, enterocolitis, etc.).

The *albuminuria* is the constant sign of such a nephritis; in degree it generally ranges between 0.1 and 0.5 gram of albumin per liter of urine, but it may become more pronounced. It persists throughout the fastigium of the infectious or toxic disorder, but as a rule passes off shortly before the beginning of convalescence. It is clinically limited to a moderate and transitory albuminuria, accompanied by very slight, temporary disturbance of the renal functions, erythrocytes, leucocytes, and granular casts appearing for a time in the sediment. Complete and permanent recovery from it is the rule, though the physician should be somewhat guarded concerning the passage of these cases of nephritis into a chronic involvement, which is, however, exceptional.

(*b*) **Typical acute nephritis** is met with under the same conditions as the preceding group, *i e*, generally in connection with and during the course of an *acute infection*, definite or obscure It is characterized by *three cardinal groups of signs* by which it may easily be recognized, and the following summary of which is adapted from Castaigne.

1. The **urinary syndrome**, which may be summarized as follows: *The urine is scanty, highly colored, comparable to* turbid bouillon, sometimes reddish, and even occasionally exhibiting hematuria. The specific gravity is high, and the reaction distinctly acid; chemical examination generally reveals a marked diminution of the urea and chlorides, together with a large amount of albumin Histologic examination of the urinary sediment shows the presence of red blood cells, leuco-

cytes, and casts of all kinds, among which the granular type is always present. The various clinical procedures recently recognized (blood-pressure, methylene blue test, and determination of the blood urea) point to a manifest impermeability of the kidneys.

2. Edema is seldom wanting in the typical forms. It is sometimes localized in distribution, as in the lower extremities, the eyelids, and even the glottis, but more often it assumes the type of a generalized anasarca with effusion in the serous cavities—pleura, pericardium, and peritoneum—and even in the viscera, particularly the brain, liver, and kidneys.

3. Symptoms due to impermeability of the kidneys are almost regularly present, but are, as a rule, limited to relatively mild manifestations such as headache, cramps, tinnitus, ocular disturbances, dyspnea, vomiting, etc.; in some instances, however, all the signs of a major attack of uremia may be witnessed, *viz.*, eclamptic seizures, acute delirium, and coma.

4. Evidences of an infectious process may be superimposed upon the foregoing symptoms, involving either the kidneys alone or the organism as a whole

The lumbar pain, sometimes very pronounced, and which may be the initial symptom, is due to an infectious process localized in the kidneys; again, palpation will reveal enlargement of both kidneys in these cases.

Infection of the entire organism is manifested in more or less pronounced fever, an enlarged liver and spleen, and leucocytosis —all showing that the infection has not been exclusively localized in the kidneys

*This is the type of albuminuria met with in typhoid fever, pneumonia, acute sore throat, scarlet fever, influenza, etc.*

(*c*) Hyperacute nephritis is usually a result of the condition of *intoxication* following exhibition of agents highly destructive to the kidneys, such as corrosive sublimate, phosphorus, cantharides, etc. The patients are previously healthy persons who have ingested one of these poisons in considerable amount and develop *almost complete anuria* on the same day; the few drops of urine collected by catheterization are found to contain much albumin and many casts

In the majority of cases, anuria remains complete in spite of all attempts at treatment and the patient dies in coma five to ten days after the beginning of symptoms, generally without having shown edema or convulsive manifestations

Thus, *anuria, coma, and death* may be said to summarize the *clinical picture,* the entire illness being gone through without the patient developing any edema or signs of advanced uremia. The ratio of blood urea may rise very high In one such case the author found 5.60 grams of urea per liter.

This applies not only to the hyperacute nephritis following *acute intoxication* in a previously healthy person, but likewise in the much more uncommon cases of hyperacute nephritis appearing during the fastigium in *acute diseases* such as typhoid fever, scarlet fever, pneumonia, etc.

Recovery is altogether exceptional. Passing mention may be made of the marked therapeutic utility of isotonic or hypertonic glucose or lactose solution in these cases.

(*d*) **Acute exacerbations in chronic nephritis.**—"These might, by the uninitiated, be mistaken for acute nephritis. The prognosis in these cases is that of the form of chronic nephritis upon which the acute congestive exacerbation has been superimposed" (Castaigne)

## CHRONIC FORMS OF ALBUMINURIA.

"The etiologic circumstances under which chronic albuminuria may be encountered are complex and should be divided into several main classes, which the physician may call to mind when confronted with a case of chronic albuminuria, *viz*, 1. *Chronic nephritis.* 2. *Chronic infections.* 3. *Chronic toxic and autotoxic states.* 4 *Circulatory disturbances.*" (Castaigne).

I. **Albuminuria of chronic nephritis.**—Albuminuria having been found to exist, it is necessary to know what variety of chronic nephritis is present and to what extent the renal functions are impaired. A systematic investigation should, therefore, be made of:

(*a*) The **elimination of chlorides,** by examining for edema and, if need be, estimation of the chloride balance

(*b*) The elimination of nitrogen, by examining for the customary signs of nitrogen retention and, in particular, by determination of the blood urea and, if need be, by calculation of Ambard's coefficient

(*c*) The elimination of water, by determination of the systolic and diastolic blood pressure and comparison of the 24-hour output of water with the pulse pressure, as well as, if need be, by calculation of Martinet's coefficient.

$$\frac{\text{24-hour output}}{\text{pulse pressure}}$$

Systematic study of these three forms of elimination leads rationally to the following classification of the chronic nephritides:

1. Simple chronic albuminous nephritis, exhibiting, apart from the chronic albuminuria, no indication of chloridemia, azotemia (nitrogenemia), or hydremia, no edema, no azotemic manifestations, and no elevation of blood-pressure.

2. Chronic chloridemic nephritis of Widal, or *hydropigenous nephritis* of Castaigne, characterized chiefly by a retention of chlorides which is clinically manifested in *edema*, without appreciable high blood-pressure or nitrogen retention.

3 Chronic azotemic nephritis— or *uremigenous nephritis,* as formerly designated by Castaigne—characterized mainly by a nitrogen retention which is manifested in a rise in the blood urea and a large number of the *classical symptoms of the uremic syndrome, viz*, headache and even rigidity of the neck, neuralgic pains, vertigo, dyspnea, general torpor, myasthenia, coma, convulsions, delirium, anorexia, nausea, vomiting, diarrhea, etc.

4 Chronic hydremic nephritis (of Martinet), or *hypertensive nephritis* (of Potain and Widal), characterized by a retention of water which is manifested in high blood-pressure, *hydremia* (anemia and lowered blood viscosity), and consequently, by predominant *cardiovascular manifestations,* such as accentuation of the second aortic sound, sometimes gallop rhythm, various forms of *hemorrhage* due to rupture of vessels (epistaxis; retinal, meningeal, and cerebral hemorrhages, etc.) followed eventually by cardiac impairment and dilatation, tachycardia, arhythmia,

loss of compensation, etc. The condition begins with cardiorenal sclerosis and leads ultimately to heart failure and uremia.

In contrast with the simple albuminous and chloridemic varieties of nephritis, the last two forms, the azotemic and the hydremic, frequently coalesce, so that, the azotemic and hydremic syndromes being in combination, the clinical picture comprises both the classic uremic symptom-complex already referred to and the equally well-known cardioarterial symptom-complex attending cardiorenal fibrosis. By combined use of the three procedures now available. 1. Determination of blood urea. 2. Of the blood-pressures, systolic and diastolic. 3. Of the extent of hydremia and anoxemia (as estimated through the blood viscosity, by refractometry, by estimation of proteins in the blood serum, etc.), it is at present possible for us to differentiate, in these ultimate complex processes, that which specially appertains to nitrogen retention from that which refers to retention of water, to anoxemia, to impaired cardiopulmonary functioning, and to cardiorenal insufficiency.

II. **Albuminuria of Chronic Infections.**—Albuminuria is very often met with in the presence of chronic infections, such as *tuberculosis, syphilis, malaria,* etc., and it is of advantage from the standpoint of therapeutic indications to establish the concurrence of a chronic albuminuria with one of the chronic infections referred to. As in the chronic nephritides, however, the prognosis is largely based, it would seem, on the functional variety of nephritis present—simple albuminous nephritis, or hydremic, chloridemic, or azotemic nephritis

III. **Albuminuria of the Intoxications.**—"In this connection," states Castaigne, "three sorts of toxic actions may be distinguished:

"The *strong* toxics (cantharides, corrosive sublimate, and arsenic in large amount) which induce acute, and in particular hyperacute, nephritis

"The weak toxics acting rapidly and not taken repeatedly (taken in a single dose) which induce a temporary albuminuria.

"The toxics taken in small but repeated amounts, and acting slowly (lead): these give rise to all the varieties mentioned in respect of the albuminurias of chronic nephritis; thus, it may

be said that, in the case of intoxications as in that of infections, etiologic data do not suffice to illuminate the prognosis."

IV. **Albuminuria of Autointoxications.**—This comprises in particular the albuminurias of pregnancy, of diabetes, and of gout.

(*a*) **Albuminuria of pregnancy.**—With Castaigne, we shall recognize a number of different varieties, which differ widely in significance and seriousness.

1. Albuminuria *of pregnant women* who already had albuminuria before pregnancy.

2. *True* albuminuria *of pregnancy*, in which the autointoxications of the latter condition are responsible for the albuminuria. The prognosis in these cases is based upon a study of the renal functions

3. Albuminuria *attending the pyelonephritis* of pregnancy, with pyuria, and the seriousness of which is dependent upon the intensity of the phenomena of retention and infection

4 The *transient and mild* albuminuria of labor.

5. *Postpuerperal* albuminuria, dependent, on the whole, upon infection, and the prognosis of which is that of the acute infectious nephritides.

(*b*) **Gouty albuminuria**—Measures should be taken to find out whether the condition is

1 A *simple albuminous chronic nephritis.* 2. A *hydremic nephritis* with hypertension, secondary to spasm of the vessels or to cardiorenal sclerosis 3 A *calculous pyelonephritis.*

(*c*) **Diabetic albuminuria.**—The significance of this form is closely analogous to that of gouty albuminuria

V **Chronic Albuminuria of Circulatory Origin.**—This type is found dependent upon two main series of causes, *viz*, cardiac and neuromotor disturbances (Castaigne).

(*a*) **Cardiac albuminuria.**—This is the albuminuria which appears at the time of attacks of acute heart dilatation or failure and seems to be dependent upon the disturbance in the return circulation, *i e*, venous stasis It generally disappears when the process of cardiac weakening ceases Where it persists, it should be studied along the same lines as were advised in the case of chronic nephritis.

| Causes | Features of the Albuminuria | Other Urinary Manifestations | Other Clinical Manifestations | State of the Other Renal Functions | Blood Examination |
|---|---|---|---|---|---|
| **Acute Forms of Albuminuria.** | | | | | |
| Mild and transient acute nephritis (infectious, subacute). | Slight, 0.1 to 0.5. Evanescent. | Hematuria, casts, both exceptional and transitory. | Blood-pressure + infection (sore *throat, grippe*, pneumonia, etc.) | Blood-pressure + transitory azotemia. | |
| Typical acute nephritis (infectious). | Moderate Lasting. | Oliguria, sometimes hematuria. Casts. | 1 Edema. 2 Signs of renal impermeability 3 Infectious phenomena (typhoid fever, pneumonia, influenza, etc.) | High blood-pressure Oliguria Chloride and nitrogen retention | Hyperazotemia. Increased blood viscosity. |
| Hyperacute nephritis (lead and phosphorus poisoning). | Abundant. | Anuria. Casts | Coma | Frequently low blood-pressure. | Hyperazotemia. Increased blood viscosity. |
| Acute *exacerbations in* chronic nephritis. | | | | | |
| **Chronic Forms of Albuminuria.** | | | | | |
| I. Chronic nephritides. (a) Simple albuminous. | Moderate and permanent | 0 | | Normal. | Normal. |
| (b) Chloridemic hydropigenous. | Frequently abundant | Chlorides reduced | Edema. | Chloride retention | Hyperchloridemia (?) |
| (c) Azotemic, uremigenous | Variable and permanent | Urea reduced. | Headache, vomiting, weakness, delirium, etc | *Nitrogen retention.* | Hyperazotemia. |

| (d) **Hydremic hypertensive.** | Small amount. | Polyuria Low specific gravity. | **Often high blood-pressure. Marked elevation of blood-pressure.** Hemorrhages, gallop rhythm, etc } Bright's disease | Retention of water. | Lowered blood viscosity (hyperhydremia). |
|---|---|---|---|---|---|

II **Chronic infections** (tuberculosis, syphilis, malaria).
III. **Intoxications.**
IV **Autointoxications** (pregnancy, gout, diabetes)
V. **Circulatory disturbances** (heart failure, dilatation of heart, vasomotor disturbances).

} The signs of the causal disorder, as well as those of one of the forms of chronic nephritis above referred to

VI **Renal hyperpermeability** (amyloid degeneration, post-*infectious asthenia*, etc) Polyuria, albuminuria, loss of weight, sometimes cachexia; occasionally glycosuria or the symptom-complex of diabetes insipidus Increase of $\frac{H}{p}$ ratio (H being the output of urine in 24 hours, and $p$ the differential or pulse pressure, estimated with the Pachon instrument) $>0\,300$.

**Cryptogenic Functional (?) Albuminuria.**

(Fatigue, digestive, cyclic, orthostatic, etc, albuminurias). Slight and intermittent } The signs of the causal disorder, as well as those of one of the forms of chronic nephritis above referred to

**Simulated Albuminuria.**

(*Pro memoriam*).—See section on **Uranalysis.**

The test for albumin in the urine should be carried out as regularly and routinely as auscultation of the chest or palpation of the abdomen.

Albuminuria is perhaps the most frequently observed of all indications of disease. Among 1000 chronic cases in city practice examined, the author found albumin 204 times, *i e*, in one-fifth of the cases; it had been overlooked in half these cases. Among 1000 acute or subacute cases (in soldiers) examined in a hospital, the author found 128 instances of transient or permanent albuminuria, *i e*, about one-eighth of the entire number.

(*b*) **Neuromotor albuminuria.**—This is the form which appears following vascular disturbances after nervous conditions such as epileptic seizures, cerebral hemorrhage, trauma to the skull, etc.

"Cases of this kind may be hard to interpret, and the physician should not be in a hurry to make a diagnosis of uremia because the patients show albuminuria consentaneously with the cerebral manifestations; only the classic tests will permit of an accurate prognosis of these combined disturbances." (Castaigne).

## CRYPTOGENIC OR "FUNCTIONAL" ALBUMINURIA.

Aside from the above mentioned groups of acute and chronic albuminuria which may be more or less readily referred to a known cause, there occurs also a relatively large number of cases of albuminuria which have not yet been completely worked out, and for which tradition has preserved the term, very probably inaccurate, of **functional albuminuria**; the term *cryptogenic albuminuria*, which mentions our ignorance of its cause without making any premature assertion as to its nature, seems to the author more rational.

The most frequent of these cryptogenic albuminurias are those known as the fatigue, digestive, cyclic, orthostatic, and intermittent and minimal albuminurias

The **albuminuria of fatigue**, which occurs intermittently and is slight in amount, appears only after prolonged, fatiguing exercise, such as hiking, running, horseback riding, etc., and generally disappears with rest.

The **digestive albuminurias** are those which arise or become accentuated during the process of digestion, whether the subjects be dyspeptic, enteritic, or normal. The relationship of cause to effect can be established only by repeated, fractional analysis of samples of gastric juice withdrawn at various stages of digestion, every precaution being taken, moreover, to eliminate orthostatic albuminuria

The **cyclic albuminurias** are those appearing in a cyclic manner, at certain periods of the day, generally between 1 and 3

o'clock P.M. Described more particularly by J. Teissier and Pavy, they seem to be dependent upon some degree of insufficiency (or debility) of the liver and kidneys.

In orthostatic albuminuria, the standing posture is the sole necessary and sufficient factor of the albuminuria, which passes off when the subject reclines. It is especially frequent in childhood.

The intermittent and minimal type of albuminuria, well described by its name, is a slight (0.1 to 0.2) and intermittent albuminuria, which appears and disappears without any sort of periodicity, independently of all fatigue, digestive process, or body posture, this constitutes the most cryptogenic of all the forms of albuminuria.

Long considered to be of a "functional" nature—*sine materia*—as well as mild, this form of albuminuria has been the subject of a long series of discussions, which have led to the conclusion that, like all the other forms of albuminuria, this clinical group may be symptomatic of a large variety of morbid states, from the mildest and most evanescent functional disturbance to the most definite chronic nephritis, and that the accurate functional diagnosis necessary for the institution of an appropriate, rational and effectual plan of treatment can be secured only by a systematic study of the renal functions (hydruria, chloruria, and azoturia) by the required methods: 1 *Hydruric balance* Blood-pressure, daily output of urine, and blood viscosity. 2. *Chloride balance:* Chloride test and examination for edema. 3. *Nitrogen balance:* Determination of the blood urea.

Mention may also be made of artificial or simulated albuminuria, of which some instances were observed during the war. Albuminuria is simulated by mixing some white of egg with the urine, either before or after urination, or even by intraurethral or intravesical injection of a solution of egg albumin Strict isolation of the suspected individual, careful watching, and collection of the specimens of urine for examination under direct supervision will readily lead to detection of the artifice. (See *Examination of the Urine* )

In this connection a final word may be said regarding the relationship of albuminuria to fitness for military service. The

(*b*) **Neuromotor albuminuria.**—This is the form which appears following vascular disturbances after nervous conditions such as epileptic seizures, cerebral hemorrhage, trauma to the skull, etc.

"Cases of this kind may be hard to interpret, and the physician should not be in a hurry to make a diagnosis of uremia because the patients show albuminuria consentaneously with the cerebral manifestations; only the classic tests will permit of an accurate prognosis of these combined disturbances." (Castaigne).

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# ALOPECIA (COMPLETE OR PARTIAL LOSS OF THE HAIRY APPENDAGES).

[ Lat. *alopecia;* from the Greek ἡ ἀλωπεχία, derived from ἡ ἀλώπηξ, the fox. ]

*"Comme il advient au regnart que son poil chiet une fois l'an, aussi est appelé le choir des cheveux allopice."*[1]

Lanfranc, folio 38, verse XIV in Littré, article *Renard.*

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Even the general practitioner is frequently consulted by patients on account of loss of hair. While not a few uncommon varieties of alopecia are very difficult to diagnosticate, even—and perhaps, especially—for the specialists, in 9 cases out of 10 the general practitioner may rapidly make such distinctions as are necessary for the application of suitable treatment.

According as the patient is a nursling, a child, an adult, or an elderly person, the diagnosis should be oriented, *a priori,* to the most frequent forms of alopecia at the patient's age.

**In the Nursling.**—In this group the condition is practically limited to:

1. **Occipital alopecia,** the result simply of wearing away of the hair on the pillow; the occiput is the area affected, and the area is ovoid in shape with its long axis directed transversely.

2 **Congenital alopecia**.—As a matter of fact, it is more particularly as the patient grows older that this form of alopecia begins to attract attention

***In the Child.***—Special thought should be given to the possibility of alopecia areata, ringworm, and cicatricial alopecia in this group of cases

1. **Alopecia areata.**—The following lines are reproduced from Sabouraud's description of this condition· "This is a primary

[1] "As it happens to the fox that his hair falls out once a year, even so is loss of the hair termed allopicia"

main conclusion from the foregoing facts is that, on the whole, albuminuria is of relatively slight, and renal functioning of paramount, importance in relation to the prognosis, and hence also in relation to military fitness. The essential point, therefore, is to ascertain the condition of renal functioning in each case. The author is entirely prepared to subscribe, with a few slight modifications, to the conclusions stated by Gilbert (*Réunion médico-chirurgicale de la Ve armée*, Oct 28, 1916): *An albuminuric subject, to be kept in the armed service, must answer the following requisites* 1 A fixed amount of albumin, uninfluenced by exposure to cold, the standing posture, food conditions, and fatigue. 2. Absence of casts. 3 Blood urea normal, and urinary urea parallel to the nitrogenous food in the diet. 4. Absence of edema, with a normal chloride balance. 5. No pronounced elevation of blood-pressure (below 180), and no gallop rhythm. A decision can be reached in such cases, therefore, only after prolonged and careful study.

at any time; on the other hand, it may extend until the entire scalp and the body surface as a whole has lost its hairy covering. Upon the scalp, extending areas of the disease are marked by the presence of the typical club-shaped hair stumps, either singly or in groups or streaks . . . Such a hair, which is suggestive of the exclamation point in ordinary printing type,

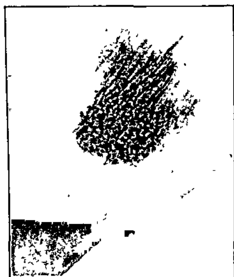


Fig 500—Microsporia (*Sabouraud*)



Fig 501.—Hairs affected with microsporia, seen with a magnifying lens (*Sabouraud*).

is one in process of atrophy; it is like a portion of a needle with its point embedded in the skin, etc."

Alopecia areata generally sets in in children between four and seven years of age and, aside from the severe and recurring forms, is spontaneously recovered from in from six months to two years

2 Ringworm.—(*a*) Tinea tonsurans due to the small-spored fungus (microsporia) is the commonest form among the tineas, *i e.*, diseases of the epidermis and hair caused by a cryptogamic

form of alopecia, which is not preceded by any functional manifestation . . . The hair is lost either diffusely over a limited

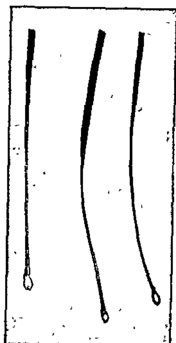


Fig. 498—Hairs in alopecia areata, viewed with a magnifying lens (*Sabouraud*)

region, or as a patch which becomes completely bald from the start . . . The bald surface is irregular in outline, of varying

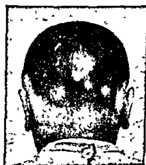


Fig 499—Alopecia areata in a child (*Sabouraud*).

shape, smooth, and devoid of any abnormal feature. It may become definitely limited or arrested, or even undergo recession,

at any time; on the other hand, it may extend until the entire scalp and the body surface as a whole has lost its hairy covering. Upon the scalp, extending areas of the disease are marked by the presence of the typical club-shaped hair stumps, either singly or in groups or streaks . . . Such a hair, which is suggestive of the exclamation point in ordinary printing type,

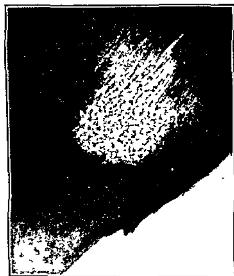


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Alopecia areata generally sets in in children between four and seven years of age and, aside from the severe and recurring forms, is spontaneously recovered from in from six months to two years.

2. Ringworm.—(*a*) Tinea tonsurans due to the small-spored fungus (microsporia) is the commonest form among the tineas, *i e.*, diseases of the epidermis and hair caused by a cryptogamic

microparasite. "It is characterized by dry, scaly, grayish patches 2 to 5 centimeters in diameter, nearly round and with rather well circumscribed margins. The very first glance at the affected area reveals that the hairs at the surface of these patches are less numerous than normally. Of these hairs, a very few have retained their normal features The others, the ringworm hairs, are short, broken off at a distance of 3 or 4 millimeters above the skin surface, decolorized, and apparently covered with

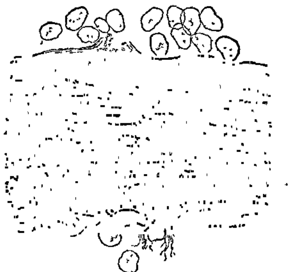


Fig. 502—Tinea tonsurans due to the small-spored fungus. Microscopic aspect of a hair (Magnified 300×). Some of the spores are seen by transmitted light (*R. Sabouraud*).

a grayish shell. The hairs thus affected break off flush with the skin when depilated. Ten or twelve grayish pieces of hair may thus be pulled out at once between the fingers. The possibility of thus depilating the surface with the fingers distinguishes this form of tinea from all others." (Sabouraud).

Microscopic examination of the hair will confirm the diagnosis.

"When heated between 2 slides in a drop of caustic potash solution and examined at a magnification of 100 to 300 diameters, such a hair reveals a cortex of very small and refractile spores, arranged in irregular apposition and forming a kind of shell

about the hair. . . . The hair appears like a little rod covered with glue and then rolled in sand."

This condition is less frequently met with in Paris than the succeeding one, and is rare in children of less than three or more than thirteen years of age

(*b*) **Tinea tonsurans due to the large-spored fungus (trichophyton or school type).**—This is, in general, less frequent than the preceding condition. When left untreated, it is characterized by numerous small areas of involvement, each of which might easily be covered by the finger-tip, and which are marked by a

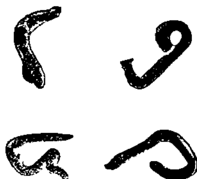


Fig. 503—Pieces of diseased hair viewed with a magnifying lens, being seen as they appear beneath the scale (*Sabouraud*).

small aggregation of adherent scales, presenting the appearance of a dry scab The diseased hair is gummed over and surrounded by the scale. In order to see it, the latter must be removed and its deep surface examined; there are then seen projecting from it little white rootlets, short and curved over Microscopic examination of these rootlets will remove all doubt Upon preparation by the technic above described, the parasite is found to consist of spores much larger than those of the preceding variety and disposed in regular series or chains and in groups of parallel, slightly wavy filaments

This variety of ringworm of the scalp, which is at present the commonest among the school children in Paris, generally occurs in girls four to fifteen years of age, though occasionally persisting to the age of sixteen or eighteen years.

(*c*) **Tinea favosa.**—In contrast with ringworm of the scalp, which is more particularly a disease of urban populations, favus is a rural variety of tinea.

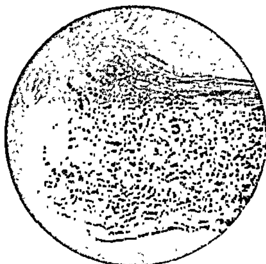


Fig 504—Diseased hair in tinea tonsurans of the large-spored variety in childhood (*Sabouraud*)

"It invades the scalp only in individuals of school age, but as it is never spontaneously recovered from, it may be encount-

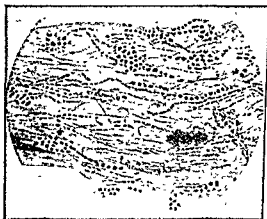


Fig 505—A hair in tinea favosa (*Sabouraud*).

ered at any period of life. In its ordinary form (*favus scutulum*) the disease is characterized by one or more irregular but sharply

circumscribed patches, covered with sulphur yellow crusts, and of the color as well as the consistency of clay. The individual, separate crusts are rounded, annular, and of all different sizes, the largest, which measure 1 to 2 centimeters in diameter, showing a series of wave-like circular elevations.

"Of these cup-like formations, the smallest form simply a ring about the hairs. The cups are pierced by the hairs and

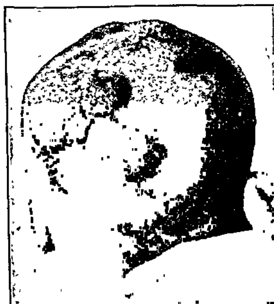


Fig. 506—Celsus's kerion (*Sabouraud*).

partly embedded in the skin They may be detached without great difficulty, in pieces; in their stead is left a bleeding wound which appears to extend rather deeply in the tissues.

"A hair affected with favus exhibits a mycelial parasitic growth composed of a few irregular, wavy, and frequently dead filaments; in the latter condition their course is shown by a clearly distinct air bubble of similar shape. The living mycelial filaments consist of segments of rather variable size and shape, with some spore-bearing portions."

(*d*) In connection with the above disorders may be mentioned certain trichophyton invasions of animal source, *e.g.*,

from the horse, dog, cat, or sheep, tending toward suppuration and leaving behind a permanent cicatricial alopecia. Among this group is the so-called kerion of Celsus.

(*e*) Under the general term **cicatricial alopecias** may be included the patches of alopecia, generally circumscribed in more or less well-defined islets, which follow *impetigo, furuncle, or trauma*. The scar-like fibrous condition of the skin in the affected area, in conjunction with the history, inevitably lead to the proper diagnosis if the case is carefully investigated.

(*f*) **Congenital temporal alopecia** is of importance only by reason of the mistakes in diagnosis (alopecia areata) to which



Fig 507—Congenital temporal alopecia (Sabouraud).

it may lead The condition occurs on either one or both sides of the head—in the latter case symmetrically—and is marked by an oval bald area 2 centimeters long and 1½ centimeters broad situated on the temple and directed obliquely upward and backward.

(*g*) The diffuse alopecias of childhood comprise particularly the **infectious and post-infectious alopecias** (typhoid fever, osteomyelitis, eruptive fevers, etc ), but are generally much less pronounced in children than in the adult.

(*h*) Lastly, mention may be made of the alopecia following **application of the x-rays.** The hair falls out twenty to thirty days after depilation with the rays, and begins to grow out again two and a half months after the exposure—unless there results an actual radiodermatitis causing, even when slight, a

permanent alopecia. A *mistake in diagnosis in this connection* cannot possibly be made. The regular, rounded shape of the bald area, suggesting a tonsure, is in itself sufficient evidence for a positive diagnosis. The *ritual alopecia of the clergy* alone exhibits such a circular shape of the bald area and the same relatively large size.

* * *

**In the Adult.**—In this group there are more particularly encountered: Seborrhea decalvans, the ordinary baldness of male



Fig 508—Alopecia due to x-ray exposure (*Sabouraud*).

neuro-arthritic subjects, the scaly alopecia pityrodes of women, and the various infectious and post-infectious alopecias, a separate place being reserved for syphilitic alopecia and various conditions suggesting alopecia areata but as yet of uncertain origin.

(*a*) **Seborrhea decalvans** (the ordinary baldness of men)—"This presents as its objective sign and fundamental lesion a cylindrical plug of fat contained in the sebaceous duct and which is caused by pressing on the skin to emerge from the duct in the form of a small vermicular mass or rudimentary comedo. This plug of fat is the seat of a bacterial colony consisting exclusively of the microbacillus of seborrhea.

"The *alopecia attending seborrhea is much less diffuse than* that of pityriasis; it is located at the vertex, over the very area at which baldness is later to result . . . As a rule, the earlier

in life seborrhea of the scalp sets in the more rapid its course. When it begins at the age of 18 it results in complete baldness at 25 years, some 200 to 400 hairs falling out each day. When it sets in at the age of 25, it results in partial baldness only at 55 to 60 years; from 50 to 60 hairs are lost a day, the number varying according to the season of the year."

Sometimes this local condition appears to be associated with

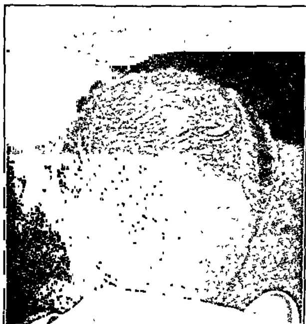


Fig. 509.—Seborrhea decalvans of the vertex Common baldness in process of development (*Sabouraud*).

or secondary to that rather indefinite, though frequently encountered diathesis: neuro-arthritism.

(*b*) **Alopecia pityrodes.**—In women this plays a rôle as important as seborrhea does in man One half of all women show some evidence of this disturbance, which should be thought of *a priori* by the physician whenever a young woman consults him on account of loss of hair. From the age of 10 years to 20 years the affected scalp is found to be scaly and covered with dandruff Later there results an elimination, not of scales and dandruff, but of the hairs themselves, which fall out whole with their bulb-like follicles and are replaced by other shorter and

weaker hairs until finally alopecia is established—never amounting, however, to complete baldness. The hair becomes more sparse, shorter, less abundant and luxuriant, with open spaces interspersed, but never with anything actually suggestive of the *seborrheic baldness of males*, with its large, regular, elliptical, smooth and polished areas of involvement.

(*c*) Infectious and post-infectious alopecia is of great practical interest. Influenza, the eruptive fevers, mumps, erysipelas,



Fig 510—Alopecia following erysipelas (*Sabouraud*).

and in particular typhoid fever, cause a more or less pronounced loss of hair "Slight alopecia may follow these conditions as soon as they have terminated, particularly those which, like erysipelas, are attended with intense local inflammation, but all of them have a definite period for the production of alopecia. *The latter follows its cause after an interval of eighty-five days. In different cases there may be five days' discrepancy, one way or the other, from this time interval.*" (Sabouraud). The hair continues to fall out for about six weeks; this occurs in a diffuse, irregular manner, without ever terminating in true alopecia. Restoration of the hair is constant in these cases.

A separate place in the classification may be set apart for the *alopeciæ of chronic tuberculous subjects, suggesting alopecia*

*areata;* this form is occipital in situation and circumferential in arrangement.

(*d*) Syphilitic alopecia is separated from the preceding group, of which it constitutes merely a *single type,* because of its great clinical importance. It appears about six months after the onset of the disease—in the course of the second six months' period, never later. "This lesion is temporoparietal and irregularly

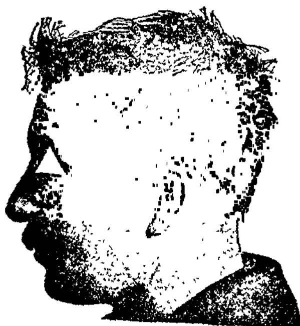


Fig. 511.—Syphilitic alopecia, rather more pronounced than usual (*Sabouraud*).

diffuse, so that when the patient's hair is cut short the hairy covering over these surfaces appears as though chopped up with poorly directed scissor cuts. At each of these points a tuft of some 12 to 15 hairs will have disappeared, leaving behind an *open space;* even in women with long hair these open spaces can still be recognized. Upon examination of the eyebrows these are found to show parallel streaks; the cervical glands are enlarged; upon looking into the mouth mucous patches are to be found. Or, general examination of the patient may reveal the indurated remnant of the chancre, the inguinal lymphatic enlargements, sometimes a still visible roseola, etc." (Sabouraud.)

Mention should here be made of the alopecia of old syphilitics and of congenital syphilitics, which resembles alopecia areata. In the congenital cases, it is accompanied by the classical stigmata—dental dystrophies, facial dystrophies, interstitial keratitis, etc. (see *Syphilis*), of the condition and will assume the form of an indefinitely protracted, recurring alopecia areata.

(*e*) Brocq's pseudopelade variety of folliculitis decalvans, on the whole rather rare, is met with almost exclusively in males 20 to 45 years of age and leads ultimately to the formation of



Fig 512.—Tinea decalvans causing almost complete baldness in a dwarf presenting all the stigmata of inherited specific infection (*Sabouraud*)

patches of alopecia measuring 1 or 2 centimeters on either side, polycyclic, serpiginous, and separated by spaces surrounded by normal hair. It exposes irretrievably more or less extensive surfaces, later coming to an end spontaneously. Its onset, course, and termination remain wholly obscure.

**In the Elderly.**—At this period of life the conditions most frequently met with are·

The advanced forms of seborrheic baldness of the adult;

A form of alopecia due to sclerosis of the follicles and representing, properly speaking, senile alopecia.

The alopecia areata of the fifties.

Cicatricial alopecia of varying origin.

*Of the first of these forms, nothing in particular need be said. Regarding the second, only slight qualifications are required.* The process of sclerosis or fibrosis leads to connective tissue replacement and disappearance of the hair follicle. The scalp, deprived of its follicles, assumes a smooth, scar-like appearance.

In *the period of the menopause,* or the process of involution taking place in women in the forties, there occurs a rather uncom-



Fig 513—Brocq's pseudopelade variety of folliculitis decalvans (*Sabouraud*).

mon parietal and frontal form of alopecia areata consisting of more or less extensive irregular patches, which are spontaneously recovered from after one or two years

*In this period of life,* finally, the scalp may exhibit, in the form of *cicatricial patches of alopecia* of varying shape and extent, the end-results of all of the foregoing possible causes of destruction of the scalp: Traumatism, furunculous eruptions, necrotic acne, cold abscesses, bone suppurations, burns, gummas, syphilitic sequestra, etc.

# ANEMIA. [a, an, *from* ἀνά, *privative;* αἷμα, *blood, no blood, little blood, poor blood.*]

As a general rule, abnormal pallor, if associated with a pale appearance of the mucous membranes of the lips, gums, and conjunctivæ, is due to *anemia.* It is a fact, however, that simple, essential, or primary anemia is very uncommon, whereas morbid pallor is of very frequent occurrence. The reason for this is that in by far the greater proportion of cases, if not invariably, the anemia is secondary to or symptomatic of some other disturbance, and that actually the diagnostic problem set before the practitioner confronted with a pale individual is that of investigating the condition which underlies the anemia.

Despite the recommendations made in current text-books, confusion of the customary pallor of anemia with the yellowish discoloration of incipient jaundice or the evanescent pallor of nervous angiospasm could occur only as a result of gross carelessness on the part of the clinician. What is more to the point, in the author's view, is that both in hyposphyxic cases and in many tuberculous patients, actual anemia may be masked by a certain amount of lividity, cyanosis, or even jaundice, particularly in hemolytic icterus. Hence, examination of the blood is, as a rule, indicated in cases exhibiting pallor. This examination should relate more especially to the cell count and the estimation of hemoglobin (see *Blood examination*). Hayem's classification is generally followed: $N$ (*number of red cells*); $R$ (*hemoglobin value*); $G=\frac{R}{N}$ (*cell value*).

*Normally,* $N$=5,000,000; $R$=5,000,000; $G$=1.

## ANEMIC STATES.

1st degree N and R, and hence also G, are very slightly reduced.

2d degree N = 5,000,000 to 3,000,000, R = 3,000,000 to 2,000,000; G = 0 80 to 0 30 (extreme figures)

3d degree N = 3,000,000 to 1,000,000; R = 2,000,000 to 800,000, G = 0 84 to 1 00

4th degree N = 1,000,000 to 300,000; R = 800,000 to 300,000; G = 0.88 to 1 70

In the 4th degree is comprised the group of the so-called *"pernicious anemias,"* the confines of which have not as yet been thoroughly determined, and which certainly occur in more than one form or variety. Determination of the differential leucocyte count is always necessary in these cases. The total white cell count is seldom increased; much more frequently the number of white cells remains normal or is diminished, in which event *leucopenia* is present. In a general way, a *plastic anemia* may be said to exist where blood repair is manifested in the appearance of young or immature cells; there may be present a leucocytosis, with granular myelocytes; nucleated red cells may be found in variable numbers, *viz.*, erythroblasts with mitotic nuclei or with nuclei in a state of pyknosis, many reds exhibiting multiple and manifest deformations, microcytes or megalocytes, etc

In the much less common condition known as *aplastic anemia,* there is an absence of defensive reaction in the bone marrow and hence also in the blood There is a leucopenia with preponderance of the mononuclear cells. Nucleated reds and myelocytes are absent.

**Anemia having been observed to be present,** the next step is to ascertain its cause, since—it cannot be too often repeated—primary, idiopathic anemia is exceedingly rare.

The classification of A. Jousset appears to the author particularly serviceable because it possesses all of the three cardinal virtues of clinical classifications, being both practical, etiologic, and pathogenetic. It combines at once the causal diagnosis and the (capitally important) diagnosis that affords rational therapeutic indications

I. **Anemias by spoliation** may follow any *traumatic, surgical,* or *spontaneous hemorrhage.* Included in this group, in particular, are all the secondary anemias attending the *hemorrhagic affections: Hemophilia, purpura, scurvy, epistaxis, metrorrhagia, hemoptysis, hematemesis, ankylostomiasis,* etc

The causal diagnosis is often self-evident in these cases. Special mention should, however, be made of the *occult gastrointestinal hemorrhages attending ulcer and cancer cases,* which require for their detection a systematic examination of the feces (see *Exami-*

THE ANEMIAS.

I.—Spoliatory Type.

1. Traumatic or operative hemorrhage.
2. Spontaneous hemorrhage in disorders attended with bleeding (hemophilia, purpura, metrorrhagia, etc.).
3. Gastro-intestinal hemorrhage, manifest or occult (ulcers or neoplasms of the digestive organs).

II.—Toxic-infectious Type.

| A. Infectious. | B. Toxic. |
|---|---|
| Chronic: | 1. Carbon monoxide. |
| 1. Malaria. | 2. Lead. |
| 2. Syphilis | 3. Mercury (?) |
| 3. Tuberculosis | |
| Acute: | |
| 1. Acute rheumatism. | |
| 2. Typhoid fever | |
| 3. Suppurative disorders | |

III.—Autotoxic Type.

1. Bright's disease.
2. Hepatic disorders.

IV.—Insufficiency of the Hematopoietic Functions.

Disorders of the blood-forming organs

V.—Cryptogenic Type.

1. So-called "primary," essential, or idiopathic anemias
2. Chlorosis.

*nation of feces; Tests for blood*) Hookworm ova are likewise detected only by examination of the stools; the patient's environment will generally afford a serviceable indication, as in miners [and in the endemic foci of the disease in the Southern U. S.—*Translator*].

**II. Anemias due to Toxic Action on the Erythrocytes.—Infectious and Post-infectious Anemias.**

In the first sub-group are placed the three major chronic infections: *Malaria, tuberculosis,* and *syphilis.* These are three of the most frequent causes of chronic anemia; if the practitioner will constantly bear them in mind he will never err when seeking the source of many chronic anemias apparently cryptogenic to a superficial observer. Should *cancerous anemia* logically be classed with the preceding forms? At any rate it should and can be classed

with them clinically, whether the anemia be due to manifest or occult hemorrhage, to toxic action on the red cells, or to both of these factors combined.

In the second sub-group are the three major acute infections: *Typhoid fever, acute rheumatism,* and the *various suppurative disorders.* In these cases the relationship of cause to effect is generally obvious.

**Toxic Anemias Proper.**—The three common forms of intoxication are those due to *carbon monoxide, lead,* and *mercury.*

The first of these is by far the most common. It may be said to be practically endemic in our cities throughout the cold season, when houses are artificially heated. The imperfect draught through most chimneys, the frequent use of heating devices under conditions of slow or restricted combustion, and the insufficient ventilation of living rooms are the effective factors in this form of poisoning. There results the well-known *carbon monoxide winter anemia* with its customary clinical manifestations consisting of dizziness, headache, and tinnitus, all refractory to any form of treatment.

*Lead anemia* comes next in frequency. The occupation of the patient (painter, plumber, etc.), the examination for other signs of lead poisoning (blue line on the gums, tremor, and high blood pressure), and sometimes the history of former attacks of lead colic, readily afford a positive diagnosis.

*Mercurial anemia* appears to the author much more uncommon, at least in his own district, if indeed it exists at all.

III. **Autotoxic Anemias.**—In this group *Bright's disease* takes first place and *hepatic disorder* comes second.

*Bright's disease* induces anemia both by causing hydremia and through an autotoxic factor. To it may be ascribed the *pallor* of persons with arteriosclerosis (the aged) and of cases of acute and chronic nephritis with or without edema. It constitutes, with cancer, by far the most frequent cause of abnormal, lasting, and progressive pallor coming on after the age of 45 In pale subjects an examination should always be made for albumin, edema, and high blood-pressure.

*Hepatic disorder* (hepatism) is, as is well known, attended with particular risk to the red blood cells; one need merely recall the time-honored and of late thoroughly illuminated conceptions as to hematic and hemolytic icterus, to realize the frequency of occurrence of such *cholemic anemias* (see *Jaundice*).

**IV. Anemias due to Insufficient Regeneration of Erythrocytes.**—Perhaps it may be justifiable to place in this group the anemias secondary to affections of the blood-forming organs: Splenomegaly, multiple adenopathies, leukemias, and bone marrow disturbances; impairment of the nervous and digestive organs with consequent poor nutrition; and polyglandular insufficiencies.

**V. Cryptogenic Anemias** of obscure or as yet unknown origin. These are the anemias that do not fall into any of the preceding groups. It is more rational to confess frankly our lack of knowledge by the term *"cryptogenic"* than to disguise it with the word "essential." *Chlorosis,* an anemia of development, appearing at puberty and disappearing at its termination, may perhaps be appropriately classed in this group until further light is thrown upon it. In this form of anemia hemoglobin reduction is more pronounced than red cell reduction, the latter often being very slight.

## APHONIA AND HOARSENESS.

[*α, privative; φωνή, sound; deprived of voice.*]

Aphonia seldom entails complete suppression of the act of phonation, such suppression being observed almost exclusively among the deaf and dumb and in hysterical mutism; it is characterized rather by *dysphonia, hoarseness,* and a muffled, raucous, discordant quality of the voice.

For practical purposes aphonia may be divided into:

*Acute* aphonia, evanescent and generally of slight import.

*Chronic* aphonia, lasting and generally of serious import.

**Acute Aphonia.**—*Acute laryngitis*.—This may be roughly divided into:

(*a*) Acute post-vocal laryngitis, practically a *traumatic* condition, occurring among speakers, actors, lawyers, "criers," vocal professionals, etc. This represents practically a "sprain" of the vocal cords.

(*b*) Acute *catarrhal* laryngitis of infections: Acute colds, eruptive fevers, measles, scarlet fever, grippe, etc.

(*c*) Acute *congestive* or irritative laryngitis, that of smokers and alcoholic subjects.

**Chronic Aphonia.**—Three main groups of causes are operative:

1. *Chronic laryngitis.* 2. *Organic diseases of the larynx.* 3. *Paralytic disturbances due to pressure upon the nerves to the larynx.*

1. **Chronic laryngitis,** the commonest causes of which are chronic fatigue of the larynx, as in criers, vendors, orators, etc., and chronic descending infections of the nasopharynx, which Laurens has aptly designated as the "morning drop of the larynx."

2. **Organic diseases of the larynx,** chiefly represented by *tuberculosis, syphilis,* and *benign* or *malignant tumors.* The clini-

cal and laryngoscopic features of these several affections will be found in condensed form in the tables on pages 686 to 690 As for their distinguishing features, the following résumé is borrowed from Georges Laurens (*Oto-rhino-laryngologie du médecin praticien*, p. 380):

"Unquestionably, for the well-trained physician, the results obtained by *auscultation* of the lungs, *bacteriologic* examination of the sputum, *specific* treatment, and *histologic* examination of a piece of tissue from the interior of the larynx may facilitate the differentiation of these laryngeal disorders, *viz.*, syphilis, cancer, and tuberculosis; the laryngoscopic picture, however, affords singularly accurate supplementary information

"The three affections are characterized by excrescences, a tumor, or ulcerations.

"(a) *Tumor or excrescences in the larynx.*

"The *syphilitic gumma* is recognized by its location, being situated in the anterior portion of the larynx, including the epiglottis and the true vocal cords; by its red, smooth, and circumscribed aspect, and by its rapid course

"*Tuberculous vegetations* are multiple, irregular, and associated with other lesions in the vicinity.

"*Cancer* results in the formation of a *single*, non-pedunculated tumor involving the vocal cord or the epiglottis, without any lesion of the adjoining mucous membrane, and *immobilizing* the vocal cord

"(b) *Laryngeal ulcerations.*

"An ulcerated *gumma* exhibits a sanious base, punched out red margins, and infiltration of the surrounding tissues.

"*Tuberculous ulcerations* exhibit dentate, irregular, and torn margins, and are multiple.

"Ulcerated *cancer* exhibits granulations and fungous outgrowths, and is sanious, bloody, painful, and unilateral."

3 Paralyses of the Larynx.—The following excellent didactic article on paralytic conditions of the larynx is likewise borrowed from Laurens's work.

## PARALYSES OF THE LARYNX.

By Dr. G. Laurens

Let the reader recall the two functions of the larynx: *Respiratory* and *phonatory*. During respiration the vocal cords move apart and the glottis is partly opened; during phonation the cords are

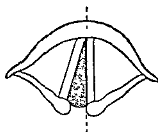


Fig 514—Paralysis of right recurrent laryngeal nerve During respiration the left vocal cord is seen to move while the right remains motionless.

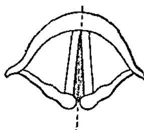


Fig. 515—Paralysis of right recurrent laryngeal nerve. During phonation the cords are seen in approximation, presenting a normal laryngoscopic picture.

approximated and vibrate, and the glottis is closed down. All these movements are carried out by the muscles of the larynx, some of which are *constrictors* (closing the glottis) and others *dilators* (partially opening it).

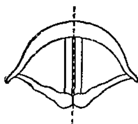


Fig 516—Paralysis of both recurrent nerves.

Within the tissue of the cords themselves are muscles (the *tensor muscles of the vocal cords*), the functioning of which insures phonation and relaxation of which causes hoarseness.

All the muscles of the larynx but one are supplied by the *recurrent* nerves. Paralyses of central, myopathic, or recurrent origin are encountered (Fig 523). The last-named variety is that occurring most frequently.

**I. Paralyses of the Recurrent Nerves.**—These may be either *uni-* or *bi- lateral*

(*a*) **Unilateral paralysis,** an aortic aneurysm, for example, exerting pressure on the left recurrent laryngeal nerve. In this instance, the corresponding vocal cord will remain motionless. The outstanding symptom is **hoarseness.**

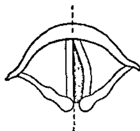


Fig. 517.—Independent paralysis of the laryngeal muscles.

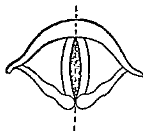


Fig 518—Independent paralysis of the laryngeal muscles.

The tensor muscles of the cords (the thyroarytenoids) are paralyzed, the cords relaxed, and the glottis presents a "button-hole" appearance.

(*b*) **Bilateral paralysis,** resulting from compression of the recurrent by a tumor of the esophagus or of the thyroid gland. This condition is rare When it occurs, the cords cannot be separated, but remain in absolute contact.

The symptoms are **hoarseness and dyspnea.**

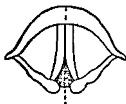


Fig. 519—Partial paralysis of the posterior muscles of the larynx (arytenoids). An isosceles triangle is formed behind the glottis

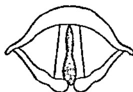


Fig 520—Paralysis of the tensor muscles of the vocal cords (cricothyroids). The vocal cords exhibit a wavy outline

**II. Independent Paralyses of Laryngeal Muscles.**—These are frequently of myopathic origin, following an attack of laryngitis, or hysterical Their presence is manifested by hoarseness.

Figures 517 to 520 show the condition of the glottis in these varieties of paralysis.

**Interpretation of a Case of Laryngeal Paralysis.**—Given a patient who has consulted the physician on account of hoarseness or dyspnea, and in whom the laryngoscopic picture has revealed laryngeal paralysis.

What data should he thereupon secure for diagnostic, prognostic, and therapeutic purposes?

*He should proceed by exclusion, proceeding from the simple to the more complex.*

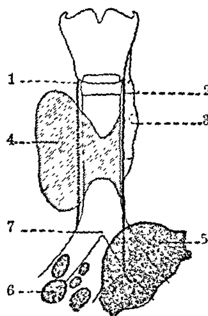



Fig 521—Diagram showing the course of the recurrent nerves, which supply the muscles of the larynx *1* Right recurrent nerve *2*. Left recurrent nerve *3*. Cancer of the esophagus *4*. Thyroid enlargement *5*. Aortic aneurysm *6*. Tracheobronchial adenopathy. *7*. Bifurcation.

*First Possibility.*—If the condition is a unilateral paralysis involving the entire cord, with the larynx otherwise normal and not in a state of hyperemia, the paralysis is one of the recurrent nerve. Let the practitioner recall his anatomy and ascertain by examination of the neck and chest which organ is causing the pressure. He should auscult, use the x-rays, the esophagoscope, etc., and will come to suspect some particular organ of causing the trouble.

Following are the possible causes of pressure on these nerves, and hence, of paralytic conditions of the larynx:

1. *Cancer of the esophagus.*—The left recurrent nerve is situated behind the trachea, in the angle between the latter and the esophagus. Consequently any growth in this organ will exert pressure on the nerve and cause paralysis of the larynx.

2 *Thyroid gland*—Enlargement of the thyroid gland, or of a *goiter,* whether developing in the left or the right lobe of the organ, exerts pressure on the recurrent nerve and paralyzes it. Such a condition may also be the result of thyroidectomy, in the course of which the nerve may be injured

3. *Aortic aneurysm*—The arch of the aorta is in close anatomic

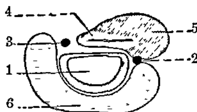



Fig. 522—Horizontal section of the neck showing diagrammatically the position of the recurrent nerves and the structures that may exert pressure on them *1* Trachea. *2* Left recurrent nerve *3* Right recurrent nerve *4* Esophagus *5* Cancer of the esophagus *6* Goiter The left recurrent nerve is thus seen to be particularly exposed to pressure from two directions.

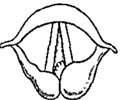
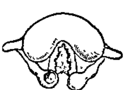
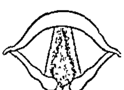
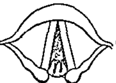
relationship wth the recurrent nerve. When dilated, it may cause paralysis of the nerve

4 *Tracheobronchial adenopathy,* developing at the bifurcation of the trachea, frequently causes pressure on the laryngeal nerves.

For clinical purposes, the greater *frequency* of left sided recurrent paralyses on account of their esophagoaortic origin should be kept in mind

Great significance attaches to this type of paralysis from the standpoints of diagnosis, pathogenesis, and treatment. Hoarseness will thus have led the physician to discover an aneurysm of the aortic arch or of the right subclavian artery. The treatment, however, is ineffectual.

| | Causes. | Clinical Signs | Laryngoscopic Examination |
|---|---|---|---|
| **I —Acute, of short duration (less than three weeks)** | | | |
| Simple, acute catarrhal laryngitis, due to overuse of the voice. | Abuse of the voice "Sprain of the vocal cords" (in orators, lawyers, criers, etc ) | Hoarseness. Constriction of the larynx. No fever. | Diffuse redness of larynx Catarrhal secretion Paresis of vocal cords. *Vocal cords roseate, catarrhal secretion; swelling of the interarytenoid mucous membrane.* *Paresis of the tensor muscles of the vocal cords (button-hole glottis)* |
| Acute congestive irritative *laryngitis* | Tobacco. Alcohol. | Do. | Do. |
| Infectious acute catarrhal laryngitis. | "Colds" Adenoiditis Eruptive diseases. Grip Herpes. | Hoarseness. Dysphagia. Fever. Symptoms of the primary disorder. | Do. + Edema of the submucous tissues, particularly at the arytenoids, exceptionally, ulcerations. Paresis of the muscles. Edema of the arytenoids. |
| **II —Chronic, of long duration (over three weeks)** | | | |
| *4.* Chronic laryngitis. | Recurring causes of acute laryngitis. | Habitual hoarseness (raw voice) Viscid, dark, rubber-like expectoration. | Diffuse redness of larynx. Redness of vocal cords Nodes on the cords. Interarytenoid thickening. Vocal cords broadened and red; thickening of the interarytenoid tissues |

| *B.* Organic diseases of larynx. | | | |
|---|---|---|---|
| 1. Tuberculosis of larynx. | Those already given + those of tuberculosis. | *Hoarseness.*<br>Inveterate cough.<br>Later, dysphagia with otalgia<br>Increasing dyspnea.<br>General signs of tuberculous disease. | 1. At the beginning:<br>Swelling of the interarytenoid mucous membrane. Monochorditis. Pallor of neighboring tissues.<br>  <br>Beginning tuberculosis.<br>Thickening and infiltration of the mucous membrane of the interarytenoid region<br>Vegetations in the interarytenoid region.<br>Thickening and red*ness of a single* cord Small interarytenoid vegetations<br><br>2. Later on:<br>Infiltration (of the epiglottis, cords, and arytenoids). Ulcers. Vegetations.<br>  <br>The established disease.<br>Infiltration of the arytenoids; interarytenoid vegetations<br>Infiltration of the epiglottis; ulcerations of the cords and of the tip of one arytenoid<br>Ulcerations of the vocal cords (glottic variety). |

| | Causes. | Clinical Signs | Laryngoscopic Examination |
|---|---|---|---|
| 2. Syphilis of larynx. | Those already given + syphilis | Hoarseness.<br>Dyspnea.<br>General condition and that of the individual organs often satisfactory<br>Syphilitic manifestations History | 1 In the secondary stage:<br>Bright red erythema<br>Mucous patches.<br>2 Later on·<br>Simple or ulcerated gumma.<br>Gumma<br>Ulcerated gumma. |
| 3 Tumors of larynx. | | | |
| (a) Benign | ? | Varying hoarseness<br>Bitonal voice. | Polyp, or<br>Papilloma, or<br>Nodule on cord.<br>Small sessile nodule on the free margin of the vocal cord.<br>Pedunculated polyp.<br>Papillomas.<br>Nodules on cords (during respiration). |

| | | | |
|---|---|---|---|
| (*b*) Malignant. | ? | Age, 40 to 60 years.<br>Increasing hoarseness<br>Increasing disturbance of respiration<br>Progressive dysphagia<br>Progressive extension of the tumor (lymphnodes, etc.). | **At the beginning:**<br>Thickening and redness *of one vocal* cord.<br>Immobility during phonation.<br>Sometimes a papillary vegetating mass.<br>(*Unilateral and immobilizing*).<br>Infiltration and vegetations immobilizing the vocal cord<br>Cancer of the larynx. Papillary vegetations.<br>**Later on:**<br>Granulating, bleeding, ulcerated, painful, and sanious tumor.<br>Extensive cancerous infiltration. |
| *C. Laryngeal paralysis.* | | | |
| 1. Recurrent paralysis; 95 per cent. of all cases. | Aortic aneurysm. | Hoarseness.<br>Signs of aneurysm. | Paralysis of the left vocal cord. |
| | Cancer of the esophagus | Hoarseness.<br>Dyspnea.<br>Dysphagia | Unilateral or bilateral paralysis. |
| | Enlarged tracheobronchial glands. | Hoarseness<br>Cough resembling that of pertussis. | Paralysis of the right or left vocal cord. |
| | *Goiter.* | *Hoarseness.*<br>Dyspnea.<br>Thyroid enlargement. | Unilateral or bilateral paralysis |

Left recurrent paralysis.

During respiration the right vocal cord moves while the left remains motionless

During phonation, the two cords being in contact, present a normal picture.

| | Causes. | Clinical Signs. | Laryngoscopic Examination |
|---|---|---|---|
| 2. Paralysis due to peripheral neuritis. | Diphtheria Alcoholism Syphilis. Diabetes. | Klebs-Loffler bacillus in exudate. Alcoholic history and stigmata. Positive Wassermann reaction Sugar in urine. | Unilateral or bilateral paralysis. Paralysis of both recurrent nerves in goiter |
| 3. Paralysis of central origin. | Spinal diseases: Tabes | Ataxia Loss of patellar reflexes Syphilitic history. | |
| | Bulbar disease: Syphilis Disseminated sclerosis Labio-glossolaryngeal paralysis, etc | The usual signs of these disorders. | |
| | Exceptional cerebral lesions· Disease of the foot of the third frontal convolution and subjacent fibers. | | |

III.—*Hysterical aphonia. Malingering.*

**1. Laryngoscopic and somatic examination negative.—***2.* **Zuber's sign** (see p. *692*).

*Second Possibility.*—If the examination of the neck and chest has given negative results, the points of origin of the recurrent nerve in the brain must be at fault Hence, the physician should make an examination of the nervous system for tabes dorsalis, disseminated sclerosis, etc. The neuropathologist will be able to put his finger on the exact origin of the paralysis —The prognosis will depend upon the cause found.

*Third Possibility* —If the hoarseness has appeared in the course of an attack of influenza or acute laryngitis; if it is an expres-

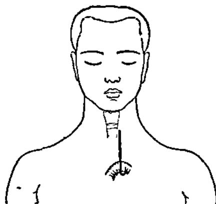


Fig 545 —The three causes of paralysis of the laryngeal muscles.

1. *Paralysis of the recurrent nerves* is by far the most frequent, any form of pressure at any point on the course of the nerve is sufficient. The course of the left recurrent nerve below, in front of, and above the aorta is shown in Fig. 545.

2 *Myopathic paralysis* is rather frequently observed It is produced as follows Following an ordinary catarrhal laryngitis the patient is seized with hoarseness This occurs from the fact that the inflammation of the mucosa has extended to the underlying tissues and set up a species of myositis The prognosis in this form of paralysis is favorable.

3 *Paralysis of central origin,* following disease of the brain and spinal cord, is much rarer

sion of isolated paralysis of one or both vocal cords, and if the mucous membrane of the larynx is still inflamed, the paralysis is unquestionably of myopathic origin. The prognosis is favorable.

**Treatment.**—This is unavailing in paralysis of central origin and very often, too, in pressure paralysis of the recurrent nerve.

It is useful in the myopathic disturbances, *i.e.*, in the paralysis following acute laryngitis; also in hysteria, in intoxications, and in syphilis.

The measures then to be recommended should comprise vocal rest, inhalation treatment (if there are still evidences of catarrh and laryngeal hyperemia), strychnine, electricity, bromides with suggestion treatment (in hysteria), and if need be, external vibratory massage In cases of cord paralysis in which the cause cannot be found the physician should not hesitate to prescribe antisyphilitic treatment, pleasant surprises for the patients cannot but thus sometimes result.

**Recurrent paralyses are by far the commonest, constituting perhaps 95 per cent. of all cases of laryngoplegia.**

The remaining 5 per cent are represented by:

**Peripheral neuritis**, as in diphtheria, alcoholism, syphilis, and diabetes

**Spinal lesions**, as in tabes dorsalis

**Bulbar lesions**, as in syphilis, tumors, softening, pachymeningitis, labio-glosso-laryngeal paralysis, disseminated sclerosis, tabes dorsalis, etc.

**Cerebral lesions** at the foot of the third frontal convolution and of the subjacent fibers.

Lastly, a word concerning **hysteria and malingering.**

Aphonia is one of the easiest symptoms to simulate, but is also one of the easiest artificial symptoms to detect, thanks to Zuber's sign When the aphonic malingerer is ordered to whistle, he insists he cannot do it; the true, non-malingering aphonic subject is generally able to whistle without any trouble, since the facial nerve is involved in this act, and is unaffected by the disorders causing hoarseness.

**ARHYTHMIA**
(*Irregularities of heart action.*)

[*a, from α, privative, ῥυθμός, measure. Irregularities of heart action.*]

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Cardiac arhythmias, manifested, in cursory clinical examination, by more or less distinct and pronounced irregularities of the pulse, constitute a very common condition. Their semeiologic significance is always pronounced and sometimes exceedingly great. The study of the arhythmias has undergone a complete renaissance in recent years, and marked clinical benefit has resulted. While certain of the arhythmias, as yet incompletely investigated, remain very difficult to interpret, the majority, on the contrary, are now well understood from the standpoint of pathologic physiology. Little mention need here be made of the as yet insoluble problems relating to these disturbances, attention being paid, as seems fitting in such a work, only to the facts that may yield useful practical deductions, *i e*, conclusions of actual therapeutic service

Careful and judicious digital palpation of the pulse, combined with correct auscultation and a critical study of the associated clinical manifestations, proves sufficient in 90 per cent of cases for proper interpretation of the more commonly encountered arhythmias. The graphic method (see the section on technic), always to be recommended where it can readily be applied, is sometimes indispensable It is the procedure of choice for the study of the arhythmias In the subjoined brief presentation of the subject, extensive and legitimate use will be made of the results secured by this procedure

In this section only a few typical examples of the cases of arhythmia most often met with in practice will be given—cases which every physician will certainly have occasion to observe, which he may rather easily discover, and with which, therefore, he should be familiar.

In practice, the most frequently encountered forms of arhythmia are:

1. *Extrasystoles* or premature contractions.
2. *Paroxysmal tachycardia.*
3. *Respiratory* (*sinus*) *arhythmia.*
4. *Auriculoventricular dissociation* or heart-block.
5. *Alternating pulse.*
6. *Perpetual arhythmia* or auricular fibrillation.

## THE NORMAL HEART RHYTHM.

In order to interpret most of these arhythmias with some degree of accuracy, a short résumé of the facts now established regarding the contraction of the normal heart may appropriately be given.

The cardiac cycle consists, as is well known, of a series of rhythmic movements, of contractions or systoles alternating with periods of rest or diastoles. The several movements constituting the cardiac cycle take place in regular succession in the following order: Auricular systole, ventricular systole, general diastole, auricular systole, ventricular systole, general diastole, etc.

All recent anatomic, physiologic, and physiopathologic investigations have tended toward the conclusion that this rhythmic succession of movements of the heart is caused by a stimulus of as yet unknown nature which, starting in the upper part of the right auricle in the neighborhood of the sinus of the superior vena cava, is transmitted from this point through the auriculoventricular septum to the bundles of muscle fibers constituting the myocardium. This apparatus for transmission of the excitomuscular impulse, or neuromyocardial propagating bundle, has been termed the *bundle of His*, after the anatomist who first described it.

The bundle may diagrammatically be conceived of as follows (Fig. 546): It originates in the *sinoauricular node*, a little mass of specialized tissue consisting of muscle cells interspersed with a rich network of nerve terminals of the cardiac nerves, and located in the upper part of the right auricle, near the opening of the superior vena cava.

The rhythmic stimulus of unknown nature elaborated in this center is transmitted along a thin neuromuscular tract, the *auriculoventricular bundle,* to a secondary center, the *auriculoventricular node,* whence it is further distributed to the myocardial bundles of each ventricle by two main branches and their subdivisions.

For practical purposes it may be said that the *systolic stimulus* originates at regular intervals of about one second in the *sinoauricular node* (the node of Keith and Flack); that it brings about contraction of the auricles at this moment, that it is transmitted along

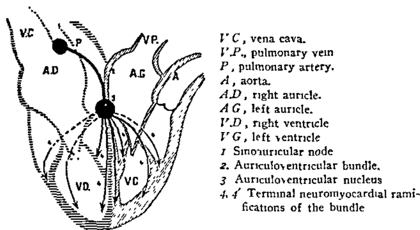
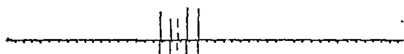



Fig 546—Diagram of the bundle which transmits the neuro-myocardial stimuli (bundle of His)

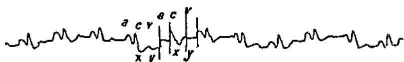
the auriculoventricular bundle to the *auriculoventricular node* (the node of Tawara), such transmission normally taking about one-fifth of a second; that it then brings about ventricular systole through transmission of the excito-contractile impulse to the myocardial muscle fibers along the branches of the above mentioned auriculo-ventricular bundle. Both auricles and ventricles then lapse into a condition of rest and lose their irritability for a period of two- to three-fifths of a second, after which the cycle of contraction above described is again reproduced.

Objective representation of this cycle is well afforded either in polygrams showing simultaneously arterial contractions such as those of the radial, which accurately register the ventricular systole with a delay of approximately one-tenth of a second

(time of transmission of the pulse wave to the radial artery), and the pulsations of the right jugular vein, which represent for practical purposes the pulsations of the superior vena cava and the right auricle; or, in a good cardiogram taken with the patient in left lateral decubitus (Pachon).



*Rj.*




*Rr.*

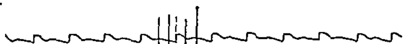
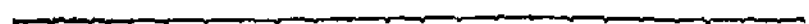


Fig 547.—Case 157 Normal pulse *Rj*, right jugular. *Rr*, right radial

A few samples of polygrams showing a normal rhythm and typical from the standpoint just referred to are herewith presented.

On a polygram the beginning of the systolic expansion of



*Rj.*




*Rr*



Fig 548—Case 504 Normal pulse. *Rj*, right jugular. *Rr*, right radial.

the radial artery is very easily located, being at start of the ascending line; if, taking into account the time required for transmission of the ventricular systolic contraction to the radial artery (about $1/10$ second), one turns from the radial tracing to the jugular tracing at a point preceding the above-mentioned

point by $\frac{1}{10}$ second, the point corresponding in time to the ventricular systolic contraction is obtained. The jugular tracing then becomes very easy to interpret (Figs. 547 and 548). Each cardiac cycle is reflected in the jugular tracing by three elevations:

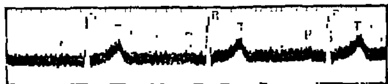



Fig 549—Normal electrocardiogram (*Daniel Routier*).

1. A *presystolic* wave, *a*, corresponding to auricular systole, and presystolic in respect of the ventricular contraction. This is generally represented by the letter *a* (auricular).

2. A *systolic* wave, *c*, immediately following the preceding, from which it is separated, as a rule, only by a very slight depression;

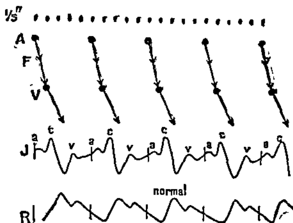



Fig 550—Diagram showing the succession of motor events in the normal heart The auricle *A* contracts first and sends its impulse to the ventricle *V* along the bundle *F* The ventricle at once begins to contract The time of transmission, which is approximately the same as the duration of auricular systole, is about $\frac{1}{5}$ second *R*, radial tracing; *J*, jugular tracing. Marks at the top Time in fifths of a second

it corresponds to ventricular systole It is generally represented by the letter *c* (carotid) because the earlier observers, Mackenzie among others, ascribed it to the carotid pulsation, which, how-

ever, does not seem always to be the case. At all events this notation will here be preserved.

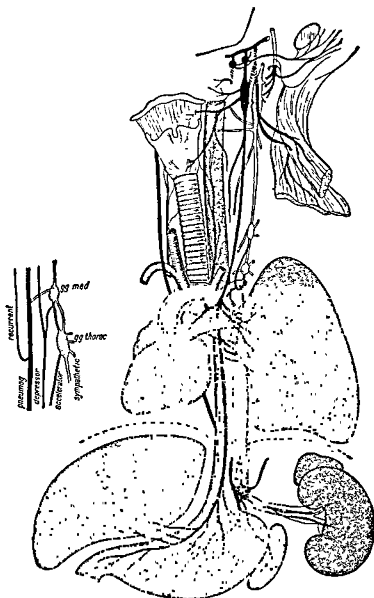



Fig 551 —The pneumogastrics (*Landois*).

3. A *post-systolic* wave, *v*, distinctly separated from the wave preceding it, *c*, and from that which follows it, *a*, in the next cardiac cycle by 2 definite depressions, *x* and *y*. This wave is

generally represented, in accordance with Mackenzie's notation, by the letter $v$ (ventricular), Mackenzie ascribing it, or at least its terminal portion, to relaxation of the right ventricle and opening of the tricuspid orifice. The exact significance of this wave has been the subject of prolonged discussion and is still being discussed; as a matter of fact it is one of the most fixed and constant and often one of the most pronounced features of the venous pulse

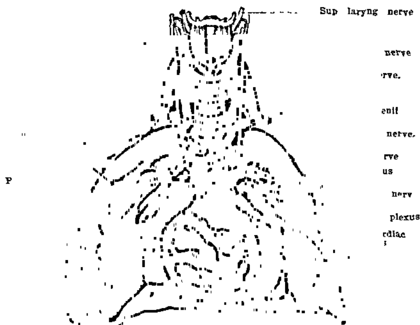



Fig 552—The nerves of the heart (*Hirschfeld*)

tracing; it corresponds practically to the diastolic rebound of the radial pulse, to the opening of the tricuspid valve and the closure of the sigmoid valves The notation $v$, is thus highly appropriate for it, provided there be attached to it the meaning *valvular*, which is more comprehensive Actually it marks the termination of ventricular systole and the beginning of general diastole of the heart.

The normal electrocardiogram lends itself to the same considerations (Fig. 549).

The successive movements of the normal heart and the transmission of the neuromyocardial impulse of contraction may be

represented in a diagram which will greatly facilitate presentation of the subject of the arhythmias (Fig. 550).

The apparatus having to do with intracardiac conduction may be and unquestionably is influenced and partly controlled by the vagus and the sympathetic. Some of the cardiac arhythmias are known to originate in the extracardiac nervous mechanism, consisting chiefly of the bulb, the vagus, and the sympathetic; it seems desirable, therefore, to reproduce and show in a diagram the distribution of these nerves (Figs. 551 to 553). These illustrations will doubtless facilitate comprehension of certain forms of arhythmia.

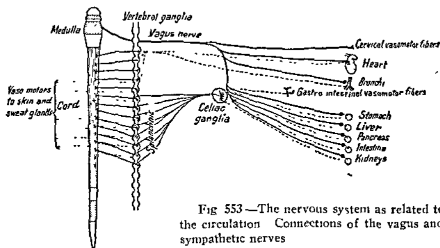


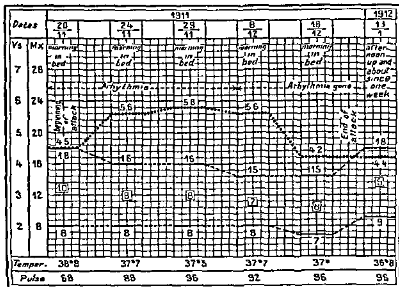

Fig 553—The nervous system as related to the circulation Connections of the vagus and sympathetic nerves

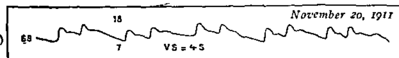
## DISTURBANCES OF RHYTHM.

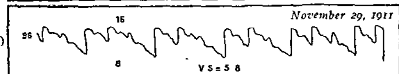
### EXTRA-SYSTOLES (PREMATURE BEATS).

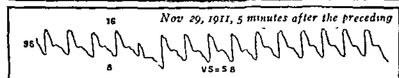
The normal cardiac rhythm is produced, we have seen, by an impulse which, starting at regular intervals from the sinoauricular node, or node of Keith and Flack, passes down along the conducting system previously described, awakening in succession a contraction of the auricle and then of the ventricle The process goes on as if the entire cardiac rhythm were governed by the primary contractions of the auricle, these in turn regularly setting off secondary contractions of the ventricle.

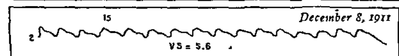
**An extra-systole or premature contraction is an extraordinary, premature systole occurring independently of the above-men-**



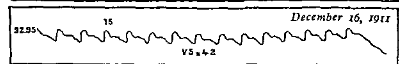




FIG. 554—Premature contractions (extrasystoles) during an attack of gout. (*a*) Bigeminal and (*b*) trigeminal pulse.

tioned regular succession of contractions. Everything takes place as if the initial stimulus arose at an abnormal point—independently of the sinoauricular node—either in the auricle, in the ventricle, or in the intermediate auriculoventricular node. Hence there are 3 kinds of extra-systoles: Auricular, ventricular, and auriculoventricular. These are sometimes rather hard to differentiate.

An extra-systole is generally felt *by the patient* as a precordial thump, accompanied by slight discomfort and an evanescent tendency to fainting.

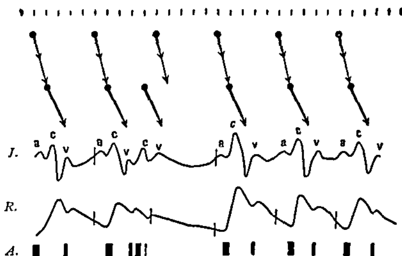



FIG. 555.—Ventricular extra-systole or premature beat. The third ventricular contraction takes place too soon. The third auricular contraction, taking place during the period of lost ventricular irritability (refractory period), fails to induce a contraction of the ventricle. *J*, jugular; *R*, radial; *A*, auscultation.

It is detected *by the physician* upon palpation of the pulse in the form of an intermittence in the latter, a pause of unusual length, suppression of one pulse wave, a "misstep of the heart." Sometimes an ordinary beat is very closely followed by a very small beat succeeded by a long pause; at other times there is felt but one ordinary pulsation, followed by a long pause.

*Auscultation yields significant results* (*Figs.* 555 and 556). If the extra-systole is strong enough (and sufficiently late in respect of the preceding contraction) to open the sigmoid valves,

the double sound of the preceding systole is at once followed by a double echo sound due to the extra-systole, and then by

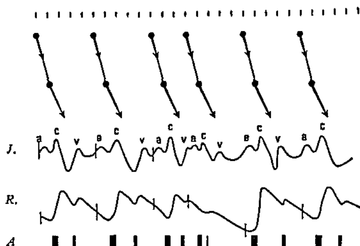



Fig. 556.—Auricular extra-systole *J*, jugular, *R*, radial; *A*, auscultation

a prolonged pause The rhythm has been reduplicated and now includes four sounds If the extra-systole is too weak (and

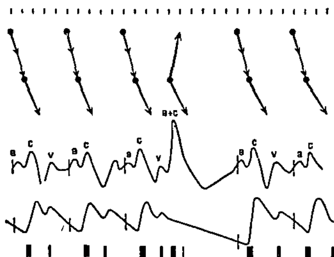


Fig 557—Auriculoventricular extra-systole

comes too soon after the preceding contraction) to open the sigmoid valve, the double sound of the preceding systole is fol-

lowed by a single sound due to the ventricular contraction in the extra-systole (the rhythm now entailing 3 sounds), and then by a long pause.

These extra-systoles may recur at quite irregular intervals, devoid of any regular rhythm. If, on the other hand, they occur in series at regular intervals they constitute *allorhythmias*. If each regular systole is followed by an extra-systole, the pulse assumes a bigeminal character; if the extra-systole recurs regularly after 2 regular systoles, the pulse is trigeminal; after 3 regular systoles, quadrigeminal, etc. (Fig 554)

Such are the simplest and most fundamental clinical observations that can be made without the help of any form of instrument

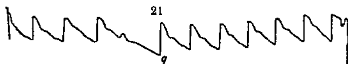



Fig 558—Case 205 Ventricular extra-systole. Pulse, 74; pressure, 21%0

The foregoing diagrams will have afforded a good demonstration of the nature of the phenomenon (Figs. 555, 556, and 557).

**Differentiation of the several varieties of extra-systoles** is attended with greater difficulty, requiring the application of the graphic method; it may even prove difficult when this procedure is availed of

Ventricular extra-systoles are distinguished from auricular extra-systoles by the 3 following features:

1. *The total* duration *of the cycle consisting of an ordinary systole and a ventricular extra-systole is equal to that of a cycle formed of two ordinary systoles; this duration is appreciably less, however, in the case of a cycle consisting of an ordinary systole and an auricular extra-systole.* This sign is the simplest, most constant, and most readily observed of the signs differentiating these 2 varieties of extra-systoles. It can be recognized even in a simple radial tracing (Fig 558)

2. On polygrams, if the extra-systole has forced open the sigmoid valves, it is shown in the tracing by a premature contraction

followed by a more or less prolonged pause, which is always definitely more prolonged than the normal diastolic pause (Figs. 558 and 559). If the extra-systole has not opened the sigmoid valves, the radial tracing shows no evidence of any premature interpolated elevation; one merely notes the absence of one beat, and a diastolic pause of manifestly abnormal duration. The jugular tracing, when distinct, is rather characteristic of one or

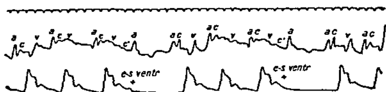



Fig. 559—Ventricular extra-systole (*Daniel Routier*).

the other variety of extra-systole. In ventricular extra-systole a rise synchronous with the extra-systolic elevation at the radial artery is observed during the abnormal pause; in auriculoventricular extra-systole, the extra-systolic jugular pulse wave often occupies exactly the place which should have been occupied by the auricular elevation, and since it brings together simultaneously the auricular and ventricular systoles, it is single and is frequently definitely higher than the normal systoles that pre-

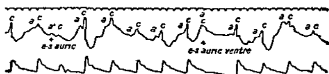



Fig 560—Auricular and auriculoventricular extra-systole (*Daniel Routier*).

cede and follow it; in auricular extra-systole, the interpolated extra-systolic jugular tracing represents in miniature the events occurring in an ordinary cardiac cycle with its three waves: *a*, presystolic or auricular; *c*, systolic or ventricular, and *v*, post-systolic or valvular (Figs. 558, 559, 560, 561).

It should not be overlooked that in some instances the tracings are rather hard to interpret; under these circumstances electrocardiography may be of service.

lowed by a single sound due to the ventricular contraction in the extra-systole (the rhythm now entailing 3 sounds), and then by a long pause.

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Such are the simplest and most fundamental clinical observations that can be made without the help of any form of instrument.

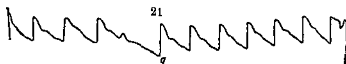



Fig 558—Case 205 Ventricular extra-systole Pulse, 74; pressure, 21%0

The foregoing diagrams will have afforded a good demonstration of the nature of the phenomenon (Figs. 555, 556, and 557).

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Ventricular extra-systoles are distinguished from auricular extra-systoles by the 3 following features:

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2. On polygrams, **if the extra-systole has forced open the sigmoid valves,** it is shown in the tracing by a premature contraction

2. Lesional extra-systoles, as a rule practically permanent, occurring in conjunction with some myocardial lesion and accompanied by the ordinary signs of myocardial and vascular degeneration which will be repeatedly mentioned hereinafter—changes of blood-pressure, stasic phenomena, dyspnea on exertion, signs of aortic degeneration, etc. In this event the extra-systole is a sign of myocardial degeneration which, taken in conjunction with the others, leads to the usual guarded prognosis of myocarditis.

In brief, detection of extra-systoles should lead the practitioner to make a complete, systematic examination of the circulatory system If the examination proves negative, the prognosis will be definitely favorable, *viz*, that of aërophagia, dyspepsia, or gout; if, on the contrary, it leads to detection of the customary signs of myocarditis, the prognosis rendered should be that of myocarditis. There is no doubt but that the extra-systole may be, in the eyes of the patient, the first, significant sign showing the presence of degenerative myocarditis—it is from this viewpoint that its detection is of such interest to the cardiologist.

### PAROXYSMAL TACHYCARDIA.

Consideration of paroxysmal tachycardia is here taken up directly following that of extra-systoles because recent cardiologic investigations have led to considering *paroxysmal tachycardia as consisting of extra-systoles, generally of the auricular type, occurring in uninterrupted succession for a period or paroxysm which may last from a few seconds to several weeks*

The subjoined diagram will satisfactorily illustrate the process and may take the place of a definition (Fig 562).

The diagnosis of paroxysmal tachycardia is relatively easy: It may be put down as an axiom that any tachycardia exceeding 110 beats per minute, sudden in onset, unaccompanied by exophthalmic goiter, not appearing in the presence of a febrile disorder, and *the rate of which is not appreciably modified by shifting from the recumbent to the vertical posture,* is paroxysmal tachycardia. Difficulty of recognition arises only in individuals seen for the first time, whose history is not known, and who, in conjunction

3. Again, whereas in ventricular extra-systoles the normal rhythm of the heart, aside from the premature contraction itself, is not disturbed, this is not true of the auricular extra-systoles, in which, even apart from the extra-systole, the heart rhythm may exhibit more or less irregularity.

The extra-systole or premature contraction is by far the most common form of arhythmia encountered in cardiologic practice. We have already seen the high degree of accuracy with which the physiopathologic diagnosis may be established in these cases On the other hand, much discussion is still going on regarding the prognosis; *the extra-systole, indeed, is a common reactive mani-*

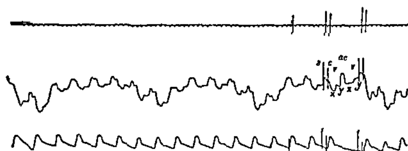


Fig 561—Case 72. Auriculoventricular extra-systole.

*festation on the part of the myocardium which may be observed under the most varied circumstances.* Dyspepsia and aërophagia frequently induce extra-systoles of reflex origin, practically devoid of significance from the cardiac standpoint; yet these same extra-systoles may be expressions of more or less marked degeneration of the myocardium.

In short, the *extra-systole per se* is of no prognostic significance; all depends upon the circulatory symptoms and signs which accompany it.

For practical purposes, there may be differentiated:

1. Functional, reflex extra-systoles (aerophagia, dyspepsia, or nervousness) or toxic extra-systoles (gout)—intermittent, temporary extra-systoles generally unaccompanied by any disturbance of the circulation save occasionally a temporary elevation of blood-pressure (neurocardiac erethism), and devoid of any prognostic significance as regards the heart and circulation

sea, and vomiting, and *cardiac manifestations*, some of the anginose type, *e g.*, distressing dyspnea or a sensation as of constriction, gripping, pressure by a tight band, or squeezing, others of the type of inadequate heart action, *e g*, congestion of the liver and lungs, cyanosis, venous engorgement, etc.

Usually the *attack ends* abruptly, as it began. Very exceptionally sudden death has been witnessed; sometimes, though very infrequently, the heart-muscle is observed gradually to give out, death taking place from asphyxia.

As in the case of extra-systoles, the *prognosis* of paroxysmal tachycardia is much less dependent upon the tachycardia *per se* than upon other attendant factors, particularly the pre-existing state of the myocardium.




Fig 563 —Paroxysmal tachycardia (*Daniel Routier*).

For practical purposes one may, as with extra-systoles, distinguish:

The functional paroxysmal tachycardia of the neurotic, the abnormally impressionable, and the sphygmolabile, unassociated with any appreciable pathological changes and exhibiting, in the intervals between attacks, a perfect circulatory balance, with absence of any permanent symptom. This form of paroxysmal tachycardia is not, as a rule, of serious import.

The organic paroxysmal tachycardia associated with or even dependent upon manifest lesions of the myocardium or endocardium, consisting most frequently of cardioarteriorenal sclerosis or of mitral stenosis The prognosis in these cases is that of the underlying disease, aggravated by a paroxysm which, by its prolonged duration, may in itself constitute a cause of rapid exhaustion of the heart-muscle

Thus, under such circumstances the author witnessed death in seven days from progressive cardiac failure in a patient 80 years old, suffering from well compensated cardiovascular-renal

with more or less pronounced tachycardia or tachyarhythmia, exhibit evident manifestations of cardiac impairment, such as dilatation of the heart, edema of the lungs, congestion of the liver and spleen, reduced urinary output, edema, etc It may be hard to find out whether the paroxysmal tachycardia was the initial manifestation of the trouble or whether, on the other hand, the tachyarhythmia observed is merely an evidence of heart failure. The sudden onset, accurate graphic studies, and the therapeutic test will settle the question of diagnosis under such circumstances.

The onset is always abrupt—and is frequently perceived by

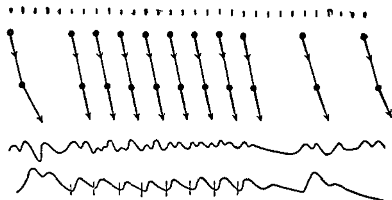


Fig 562—Diagram representing a brief attack of paroxysmal tachycardia consisting of eight successive auricular extra-systoles (premature contractions) A ventricular contraction takes place in conjunction with each of these extra-systoles. Note the abrupt onset and termination of the attack and the unusual prolongation of the final pause

the patient as a kind of sudden thump in the precordium, a sensation of unleashing of the heart, or a pronounced palpitation coupled with general malaise;—at times, however, no subjective *sensation is awakened.*

The **duration** may be extremely short—the attack consisting merely of a series of premature beats varying from a few to several dozens in number. The attack generally continues from a few hours to a few days, more rarely a few weeks.

Sometimes the **paroxysm** is unaccompanied by any appreciable subjective *sensation.* Usually, however, there are observed *digestive disturbances,* such as flatulence, regurgitation, nau-

an inhibitory effect upon it. Destruction of the pneumogastric, more especially of the trunk on the right side, or its physiologic suppression by the administration of atropine, which paralyzes




Fig 564.—Case 236 Respiratory (sinus) arhythmia.

it, consequently accelerates the heart-rate; stimulation of it, on the other hand, slows the heart.

As a rule, in man, this brake-like or inhibitory action of the vagus is not noticeable. In certain individuals, however, par-

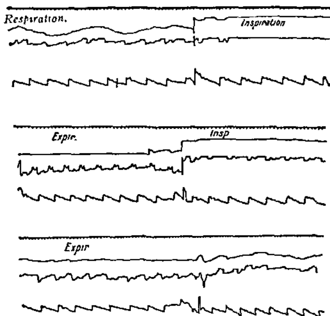



Fig 565.—Case 236 Sinus arhythmia.

ticularly in the majority of children, as well as in a few adults (and always in dogs), this action is plainly present and is manifested in a pronounced arhythmia, affecting both the frequency

sclerosis, who was seized with paroxysmal tachycardia (heart-rate 170 to 180) one evening. Dyspnea was rather pronounced, the respiratory rate ranging from 22 to 48; the heart-sounds were muffled and unequal; no cough, no fever, no expectoration, no edema, and no congestion of the liver or lungs; there were marked borborygmi and meteorism, and dyspnea and arhythmia recurred upon the slightest attempt to take food; the least exertion brought on a feeling of constriction, pressure, and oppression, with anginose manifestations.

Although not a complete failure, treatment by mustard packs, digalen, camphor in oil, sparteine, oxygen injections, etc., proved insufficient. Progressive weakening of the heart action was witnessed, with associated congestion of the bases of the lungs, paroxysmal seizures of cardiac dyspnea, reduced urinary output, and polypnea. Death supervened on the seventh day.

The author's other cases of the same disorder recovered after paroxysms lasting from a few hours to a few weeks.

In short, the pathological lesions present along with the syndrome, and the duration of the tachycardial attack, are the main factors governing the prognosis. Generally the prognosis as regards continuance of life is favorable, even in the presence of advanced sclerotic lesions.

## RESPIRATORY (SINUS) ARHYTHMIA.

**Respiratory** or sinus arhythmia is, next to extra-systolic arhythmia, the form of heart irregularity most frequently encountered in practice It is definitely known to be the mildest of the several forms of arhythmia.

The description of the normal heart rhythm already given will greatly facilitate comprehension of this form of arhythmia. In accordance with the physiopathologic observations already recalled, the normal rhythm of the heart is dependent upon a stream of regular stimuli starting rhythmically in the sinoauricular node (node of Keith and Flack) and transmitted thence in succession to the auricular myocardium and the ventricular myocardium by the conducting system previously referred to. This sinoauricular node, however, is itself manifestly under the control of the vagus or pneumogastric nerve, which exerts

respiratory movements are purposely amplified to the point of deep inspiration and forced expiration.

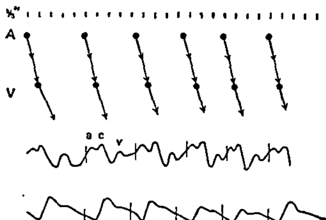



Fig 568—Diagram illustrating respiratory (sinus) arhythmia. *A* and *V* stand, respectively, for the auricular and ventricular contractions, taking place in normal succession The arhythmia here consists of an alternate acceleration and slowing of the auriculoventricular cycles, due to actual arhythmia of the initial stimulus originating at the sinus

This form of arhythmia, representing at most the exaggerated expression of a normal process, perhaps points to an in-

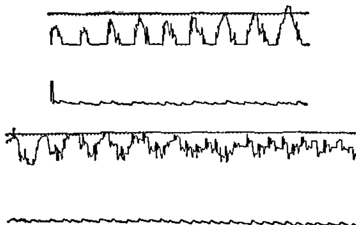


Fig 569—Case 36 *ter*. Cheyne-Stokes rhythm H, 61 years; Feb 28, 1913, sitting position; pulse, 100 (?), pressures, 340/180, viscosity, 6.4

creased irritability of the sinoauricular node. At all events it is certainly devoid of all prognostic significance, and yields

and the amplitude of the heart-beats, and distinctly subordinate to the respiration. Even a cursory examination, with simultaneous palpation of the radial pulse and the respiratory rhythm (inspiration and expiration), reveals a manifest relationship be-

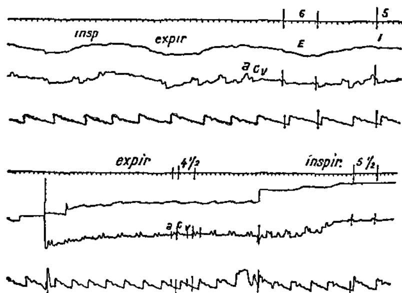


Fig 566.—Case 236 Sinus arhythmia

tween the arhythmia and the time of the respiratory movements, the circulatory irregularity being observed to consist of an acceleration of the pulse occurring with inspiration and a slowing of the pulse with expiration.

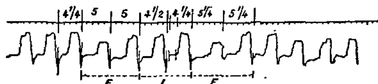


Fig 567—Case 263 Sinus arhythmia Cardiogram
*E*, expiration, *I*, inspiration.

The tracings herewith reproduced (Figs 564 to 567) clearly illustrate this close interdependence between the circulation and respiration. As a matter of fact, the phenomenon is merely the expression of a normal condition to an exaggerated degree, and may almost always be recorded if, while a tracing is made, the

tricular dissociation of the first degree, or better, a tendency toward heart-block (Fig. 572).

If transmission is interrupted from time to time, if some

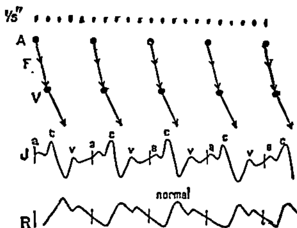


Fig 571.—Normal tracing

obstacle to conduction results in its being occasionally broken off, *some of the auricular systoles will fail to send their contractile impulses to the ventricle.*

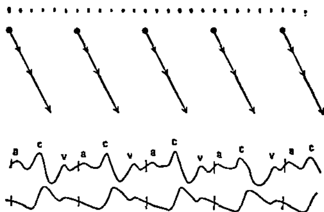


Fig 572.—Tracing showing a tendency to auriculoventricular dissociation. Delayed conduction. Prolongation of the *a-c* interval

Suppression of some of the ventricular contractions will occur *under these conditions* This *constitutes auriculoventricular* dissociation of the second degree or incomplete (partial)

but a single therapeutic indication, *viz.*, that of allaying all apprehension on the part of the patient and his family, and therefore of ordering no treatment, which might simply lead the patient to fear and believe that some abnormal condition was really present in his case.

The above diagram will assist the reader in understanding the probable mechanism of this form of arhythmia (Fig 568).

As a matter of interest there is also reproduced herewith a tracing from a case of respiratory arhythmia in which conditions were manifestly far different from the group previously referred to, the tracing was obtained in an azotemic patient during an attack of cardiorespiratory dyspnea of the so-called "Cheyne-Stokes type" (Figs 569 and 570).

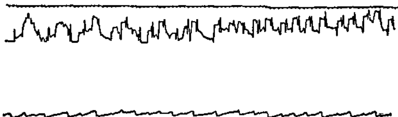


Fig. 570—Case 36 *ter* Cheyne-Stokes rhythm (*continued*). (To be read from right to left)

## AURICULOVENTRICULAR DISSOCIATION.

The diagrams already presented in illustration of the normal heart rhythm, extra-systoles, and paroxysmal tachycardia are of further marked utility in defining and describing *auriculoventricular dissociation* or *heart-block*

The normal rhythm of the heart is dependent upon regular transmission of a stimulus to contraction from the sinoauricular node (of Keith and Flack) from the auricle to the ventricle along the bundle of His (Fig 571)

If this process of transmission is protracted or delayed owing to some hindrance to conduction, as shown in Fig 572, a tendency to heart-block will occur which will be shown in tracings by increased length of the *a-c* interval and by the appearance of a short pause between the end of auricular systole and the beginning of ventricular systole. This constitutes auriculoven-

in a certain number of instances One is therefore led to the conclusion that, as in the case of extra-systoles or tachycardia, there may occur, aside from the organic, permanent auriculo-ventricular dissociation due to disease of the bundle of His (gumma, fibrosis, post-infectious or post-rheumatic degenerations), cases of functional, transitory auriculo-ventricular dissociation (digitalis block, temporary block in rheumatism and infectious diseases, stimulation of the vagus, etc.).

For practical purposes, syphilis, rheumatism, and connective

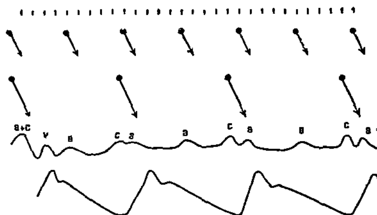



Fig 574—Complete auriculoventricular dissociation The auricles and ventricles contract inco-ordinately and independently.

tissue degenerations are dominant in the etiology of auriculo-ventricular dissociation

Finally, it may be added that, in regard to the semeiology of bradycardia, it is necessary to distinguish instances of *bradycardia* which might be termed *fascicular,* being due to disease or defective functioning of the bundle of His and auriculoventricular dissociation, and the *nodal* or *total* forms of *bradycardia,* the result of deficiency in the process of stimulus production in the sino-auricular node, and unaccompanied by auriculoventricular dissociation

**Diagnosis**—The problem of *diagnosis of auriculoventricular dissociation,* as it is placed before the unspecialized practitioner, may seemingly be presented as follows:

heart-block (Fig. 573). If the interruption, at first accidental and irregular, increases and becomes regular and rhythmic, the degree of dissociation may be expressed in some definite ratio. Thus, if the ventricle responds once out of twice to the auricular stimulus, the heart-block is stated to be of the 2:1 variety; if it responds only once out of three times, of the 3:1 variety, and so on.

The **ultimate degree of auriculoventricular dissociation,** or **complete heart-block,** is produced when all conduction between

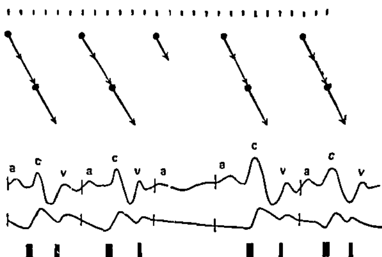



Fig. 573—Partial heart-block Incomplete auriculoventricular dissociation.

the auricle and ventricle is arrested, as illustrated in Fig. 574; the auricles and the ventricles contract independently; their respective rhythms are completely dissociated and unrelated. The auricular rate is about 72 to the minute, while the ventricular is 30. There is said to be present a bradycardia through auriculo-ventricular dissociation.

The foregoing statements constitute the simplest description that can be given of auriculoventricular dissociation or heart-block (Figs. 571 to 574).

The seat of this form of arhythmia is plainly the bundle of His. This bundle has been found diseased in most of the cases of auriculoventricular dissociation coming to autopsy; yet this systematic investigation has given absolutely negative results

2. *Is the dissociation functional in type* (usually of extra-cardiac origin and due to abnormal excitation and defective functioning of the pneumogastric nerve)—*or is it organic* (of intracardiac origin and due to a syphilitic or rheumatic fibrotic disease of the bundle of His)?

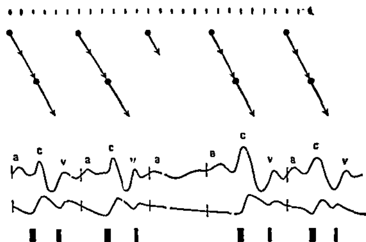



Fig 576—Partial heart-block

Auscultation will promptly answer the first question, as it did in the case of extra-systoles Combined palpation of the radial artery and auscultation will show that for every radial pulsation noted there are two contractions of the heart—one a systole and the other an extra-systole

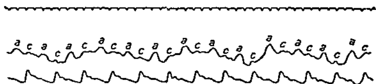


Fig 577.—Delayed conduction (*Daniel Routier*).

In answering the second question the following clinical features should be availed of.

1. Functional bradycardia (of extracardiac origin) is, as a rule, transitory, terminating upon cessation of its original cause; organic bradycardia (of intracardiac origin), however, is permanent.

The *diagnosis of* incomplete auriculoventricular dissociatio is practically limited to a differentiation of this condition fror *extra-systole.* Radial palpation and simultaneous auscultation o the heart permit of easily and almost positively settling th question. In auriculoventricular dissociation, the pause note at the radial artery is coupled with complete absence of heart sounds, since no ventricular contraction takes place (Fig. 573) in extra-systole, on the other hand, the pause at the radial i coupled with one or two heart-sounds due to the superadde

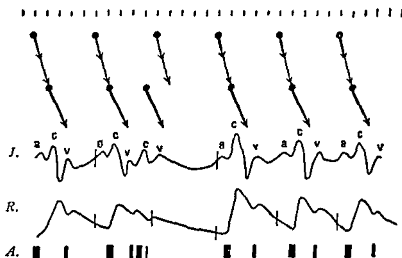


Fig. 575.—Extra-systole (premature contraction).

extra-systolic contraction of the ventricles (Fig. 575). In the first instance there is present the ordinary rhythm merely slowed down in two phases, or rather, consisting of the two sounds, systolic and diastolic; in the second instance there is a three-phase rhythm comprising the two normal systolic and diastolic sounds followed by the systolic sound of the extra-systole, or a four-phase rhythm (echo rhythm) if the extra-systole, having forced open the sigmoid valves, is accompanied by a second, diastolic sound (Figs. 575 and 576).

*The diagnosis of* complete auriculoventricular dissociation *which is objectively manifested in a very pronounced bradycardia* (30 to 40), *is made as follows:*

1. *Is there present bradycardia or bradysphygmia?*

577, 578, 579, 580).[1] Electrocardiography will almost certainly settle the question when availed of as a last resort in doubtful cases (Fig. 581). The most characteristic indications in the tracing are:

1. Abnormal prolongation of the *a-c* period
2. A pause—which may be very brief—between the end of *a* and the beginning of *c* in cases of incomplete dissociation.
3. Dissociation of the auricular rhythm *a* in the jugular tracing from the ventricular rhythm (radial pulsations and apex-beat) in cases of complete dissociation.

It should be noted that in the complete form of dissociation *there is often perfect adaptation of the system to the new circulatory régime,* the slowing of the systolic contractions being com-

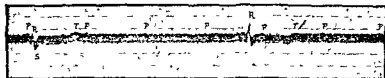


Fig 581—Complete dissociation Electrocardiogram (*Daniel Routier*). 52 11 12 Aur = 62, ventr. = 17 Lead I, ord 1 cm. = 1 millivolt, absc. 2½ cm = 1 meter

pensated for by their increased power, objectively manifested in the resulting full, large pulse—very striking upon palpation and in the tracings—and in an increase of both the systolic and diastolic blood pressures The ventricle, well filled in the course of a prolonged diastole, empties itself completely as the result of a powerful contraction

Proper diagnosis of auriculoventricular dissociation is a matter of great prognostic and therapeutic importance When functional, it is unattended with risk, as a rule, and terminates as soon as its original cause is removed When organic, it points either to serious myocardial degeneration, of which it constitutes in itself an especially dangerous local manifestation, or to the localization of some destructive or degenerative process, such as

[1] For further details the reader is referred to Routier's thesis entitled "*Étude critique sur les dissociations auriculo-ventriculaires*" Paris, J. B. Baillière, 1915.

2. Administration of 2 milligrams of atropine, by paralyzing the cardiac terminals of the vagus, will generally cause functional bradycardia *to disappear for a time; organic* bradycardia, on the other hand, is not perceptibly influenced by it.

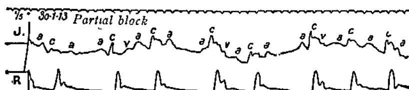



Fig 578—Partial heart-block (*Daniel Routier*).

3. Change of posture (*e.g.*, passing from recumbency to the erect posture and *vice versa*), exertion, deep or forced inspiration, fever, and locomotion cause a distinct change in the rate in

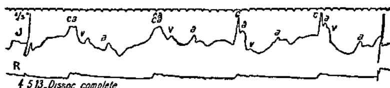



Fig 579—Complete dissociation (*Daniel Routier*).

functional bradycardia; in organic bradycardia, on the other hand, they exert no appreciable effect.

Many recent investigations have seemed to show that these indications are not of absolutely positive significance

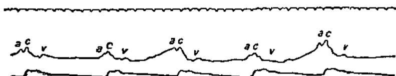


Fig 580—Total bradycardia (*Daniel Routier*).

For the practitioner specializing in cardiology, the problem of diagnosis is often markedly facilitated and illuminated by the employment of graphic procedures, whereby auriculoventricular dissociation may be recorded in the jugular, radial, and cardiac tracings, as shown in the annexed diagrams for which the author is indebted to his colleague, Daniel Routier (Figs.

tole causes in some subjects to the serious syncopal attack with prolonged cardiac arrest which may result in death.

Before Charcot's time such a condition was ascribed mainly to the myocardium, which had sometimes been found in a state of degeneration; in accord with Charcot's view, it was attributed chiefly to the extracardiac nervous system (medulla or vagus

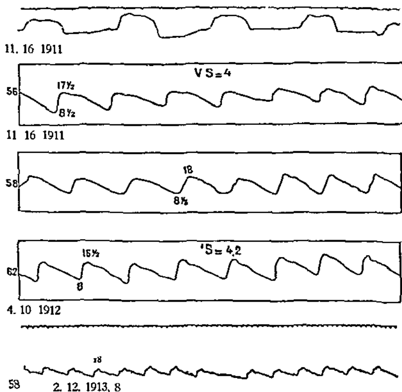



Fig. 583—Case 263 *bis*. Cardiogram and sphygmogram taken at different stages in the course of the case referred to in Fig 582

nerve); after the discovery of heart-block, however, it came to be ascribed wholly to auriculoventricular dissociation, due, in turn, to disease of the bundle of His. At the present time there exists a *tendency to revert toward a much more eclectic* conception of the condition, according to which the Stokes-Adams syndrome—slow pulse with syncopal or epileptiform attacks—may be caused by any functional disturbance or any organic

syphilis, rheumatism, or some infectious disorder, in the bundle of His.

*The study of auriculoventricular dissociation, and more particularly of bradycardia, is intimately related to that of* Stokes-Adams' disease. The features presented in this condition are well known. The patient suffering from it is subject to syncopal,

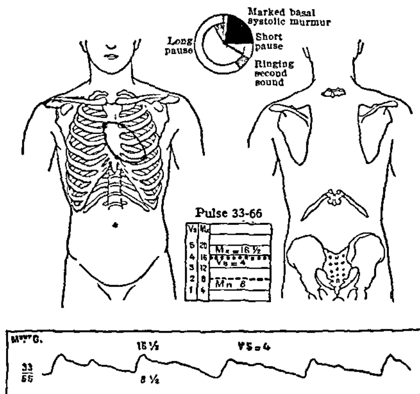



Fig. 582.—Oct. 16, 1911. Case 263 *bis.* H., 59 years. Bradysphygmia *counteracted by belladonna. Marked dilatation of aorta Cardiac* hypertrophy. Elevation of the subclavians.

epileptiform, or syncopo-epileptiform attacks. The attack is heralded by a kind of aura, characterized by general malaise, tinnitus aurium pallor of the face, etc.; it is associated with a paroxysmal slowing of the pulse, with pauses—actual periods of asystole in the literal sense of the word—which may continue for ten or more seconds. The disorder may be present to any extent and with any degree of frequency, varying from the transient mental confusion similar to that which an extra-sys-

heart-beats were double as compared to the pulse (66). Auscultation elicited the characteristic "echo rhythm"—Toc Toc, *toc toc.* The radial tracing was typical, showing a bigeminal pulse the result of extrasystoles, with the second pulsation impalpable, producing the condition termed *bradysphygmia.* The systolic blood pressure was 165 mm., the diastolic, 85 mm., and the blood viscosity, 4 No albumin

In short, there were present *aortitis and myocardial degeneration, bradysphygmia due to bigeminal extrasystoles,* and *syncopal attacks.*

The author merely prescribed, along with a liberal diet, the following pills:

| | |
|---|---|
| Extract of belladonna | 0 01 gram. |
| Extract of adonis | 0 10 gram. |

To make one pill —Five pills a day.

Almost immediately the dizziness and malaise passed off, and the pulse rate rose to 60 through disappearance of the extrasystoles. The blood pressure rose slightly to 175 to 180 mm, systolic, and 85 or 80 mm, diastolic The patient gradually resumed his former occupations. His restored condition has now been maintained for six years

In a second case (No. 164*bis*), encountered in a patient 54 years of age, likewise having an aortic lesion and suffering from attacks sometimes of a syncopal and at others of an epileptoid type, with marked slowing of the pulse, tracings made in the intervals between attacks showed no abnormality of heart rhythm save a slight tendency to slowing of the rate (58), with practically normal blood pressure (145 and 95 mm.) and a slightly increased viscosity (4 5)

Prognosis.—The *prognosis* depends:

1. On the nature of the dissociation: If functional, it is generally not of serious import and terminates when its toxic, diathetic, or infectious (rheumatism, pneumonia, or typhoid) cause is removed.

2. On the grade of the heart-block: Manifestly the slight, partial, and temporary grades of block are less serious than the pronounced, complete, and permanent forms.

change capable of inducing a pronounced slowing of the pulse-rate:

1. *Organic disease or functional disorder of the medulla or of the pneumogastric.*

2. *Organic disease of the bundle of His and at times more particularly of the node of Keith.*

3. *Or even extensive and severe disease of the myocardium without any special localization in the organ*

At all events, the author has encountered Stokes-Adams' disease in two cases in which auriculoventricular dissociation seemingly could with justification be excluded.

In the first of these cases (Figs. 582 and 583), the patient was a man aged 59 years, suffering chronically from rheumatism and with a past history of jaundice, exhibiting an extensive aortic lesion and a marked systolic murmur at the base of the heart, succeeded by a rolling sound which continued throughout the short pause, and who for several years previously had noticed a slow pulse (55) in the morning on awakening. For the preceding seven or eight months this patient had been subject to periods of dizziness and confusion consentaneous with a slowing of the pulse to 48, for the last six months he had been subject to sudden, alarming, quasi-syncopal attacks with sudden pallor and transient amnesia which had compelled him to give up completely his ordinary occupation. Having been obliged at the time to stay abed for seven weeks because of complete inability to rise without syncope, he had found that when recumbent he felt very well and was in complete possession of his mental faculties, whereas if he sat up he felt faint and was completely amnesic A strict diet and treatment with a combination of adonis and theobromine had made him, if anything, worse; a more generous diet had enabled him to leave his bed, and a stay in Savoy brought about some general betterment Bradycardia (?) and syncopal attacks continued, however, and it was under these circumstances that the patient came to consult the author The man showed an extensive and manifest aortic lesion (pronounced systolic murmur continued as a rolling sound during the short pause, and a clanging sound in diastole). The pulse rate was remarkably low (33), but the

being an expression of deep-seated myocardial degeneration, is considered by Gallavardin as "a highly important sign—perhaps the best sign—of inadequacy of the left ventricle," while the latter points to auricular fibrillation.

The alternating pulse consists essentially in the alternate occurrence, at practically normal, regular, and equal intervals, of a large beat and a small beat. There is strictly speaking, no arhythmia, but simply a regular alternation of two unequal beats. At the most, the small beat may be slightly delayed due to a slight retardation of cardio-peripheral conduction.

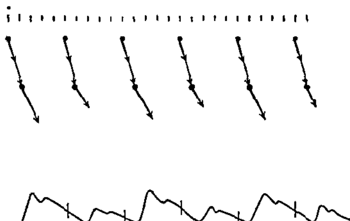


Fig 584—Diagram of the alternating pulse Strong and weak contractions take place in alternation

A bigeminal pulse the result of extrasystoles might possibly be confused with the alternating pulse, being likewise attended with an alternation of large and small pulse beats. In contrast with the alternating pulse, however, the extrasystole or small systole comes closer to the preceding than to the following beat; in the alternating pulse, the small beat is closer to the succeeding than to the preceding beat. If careful palpation and auscultation fail to settle the question, an ordinary sphygmographic tracing will promptly do so.

True alternation of the pulse is of the gravest prognostic import, and Lewis does not hesitate to compare it with subsultus tendinum, optic neuritis, and risus sardonicus as a sign of the most unfavorable portent. Frequently it is associated

3. On the other myocardial or endocardial lesions simultaneously present: The prognosis in complete, permanent auriculo-ventricular dissociation is grave, in the first place because it is an expression of a particularly serious localization of the myocarditis, and secondly and more especially, because it is usually accompanied by deep-seated and extensive myocardial degeneration.

4. On the syncopal and epileptiform attacks which may accompany the condition

(*a*) In the milder form, there are noted brief fainting spells with transitory unconsciousness, facial pallor, and a very short period of pulselessness.

(*b*) In a more advanced form, the unconsciousness is supplemented by convulsive movements of the face and upper extremities. It should be noted that incontinence of urine and biting of the tongue are, as a rule, absent. The condition of the circulation at the time is characterized by absence of the ventricular contractions, resulting, in turn, in pulselessness, and by persistence of the auricular contractions, manifested in rapid wave-like movements along the veins of the neck.

(*c*) Death may be observed—though exceptionally—in the course of an attack or a series of attacks of the type above described

## ALTERNATION OF THE PULSE.

None of the forms of arhythmia previously referred to, *viz.*, extra-systoles, paroxysmal tachycardia, sinus arhythmia, and bradycardia, in itself supplies any definite prognostic indication. The fact was sufficiently stressed that these disorders do not, taken alone, constitute accurate prognostic factors, and that each of these varieties of arhythmia is of markedly variable significance according as it is of a functional or organic nature, and that, on the whole, the condition is of clinical value and significance only as a factor taken in conjunction with the other existing disturbances. This is by no means the case, however, with the two forms of arhythmia still to be described, *viz*, the alternating pulse and perpetual arhythmia, to both of which attaches a definite and serious clinical signification. The former,

servation of the muscle surface reveals its extreme and incessant activity, rapid and minute twitchings and undulatory movements are visible over the whole. It is believed that the tissue mass has suffered functional fragmentation and that a number of small areas give independent birth to new impulses. . . . The effect of the auricular confusion upon the ventricle is two-fold. The normal, regular and co-ordinate contractions in the auricle are in abeyance and consequently the ventricle is robbed of the regular impulses which form its accustomed supply. These are replaced by numerous and haphazard impulses, escaping to the ventricle

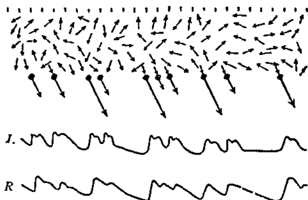



Fig. 585.—The auricular muscle fibers fail to contract in co-ordinate and rhythmic fashion The auricular tissue is dissociated into a large number of small areas contracting independently. Some of the auricular impulses reach the ventricle at wholly irregular intervals, awakening there contractions which are both frequent and irregular. *J*, jugular; *R*, radial

from the turmoil which prevails in the upper chamber; the change in the action of the ventricle, when the auricle fibrillates, is consequently profound Its rate of beating rises considerably and the contractions follow each other in a completely irregular fashion."

The above diagram will give an approximate idea of the mechanism involved (Fig. 585)

Electrocardiography plainly demonstrates the fact that the mechanism above described is actually operative. The *P* wave characteristic of auricular systole is suppressed and is replaced by a

with cardiac dyspnea, anginal attacks, and Cheyne-Stokes breathing. But even when present alone, its sombre prognostic import is retained. It is a certain indication of deep-seated myocardial degeneration and of advanced exhaustion of the heart-muscle.

A few recent observations, those of Gallavardin among others, have seemingly tended toward reduction of the gravity of the prognosis in these cases The author has personally observed three distinct cases of alternating pulse with respective survival periods of six, seven and a half, and fifteen months.

## PERPETUAL ARHYTHMIA (AURICULAR FIBRILLATION).

**Perpetual arhythmia**, the *delirium cordis* of olden times, was for a long period a puzzle to cardiologists To electrocardiography we owe, if not an absolute, complete elucidation of the condition, at least an explanation which accounts for the great majority of cases and affords the best view into its mode of production.

Perpetual arhythmia consists, as the term implies, of a permanent arhythmia characterized by extreme irregularity and baffling all description. The succeeding systolic contractions are irregular both as to duration and intensity.

Prolonged discussions as to the exact pathogenesis have been indulged in;—electrocardiography seems to have definitely shown that this type of arhythmia is dependent upon a special condition of auricular activity which is well expressed by the term **auricular fibrillation.**

The following description of this condition is borrowed from Thomas Lewis: "When we inspect the normally beating heart of an animal, the systoles of both auricle and ventricle are readily discerned. The movement of the auricle is a sharp flick, most clearly perceptible in the length of the auricular appendix, for in this line the shortening is greatest. When the auricle is forced into fibrillation or delirium, the appearances are quite distinctive; the muscular walls are maintained in a position of diastole; systole, either complete or partial, is never accomplished; the structure as a whole rests immobile; but close ob-

2. *Any persistent arhythmia* coupled with signs of advanced cardiac impairment is almost always dependent upon auricular fibrillation. The probability becomes practically a certainty if the arhythmia is associated with tachycardia.

3. *Any case of arhythmia,* even if unaccompanied by manifest signs of existing cardiac impairment, any arhythmia which is increased by acceleration of the pulse, such as might be induced by moderate exercise, is likely to be a perpetual arhythmia. The



Fig 587 —Auricular fibrillation (*Daniel Routier*).

other types of arhythmia, on the other hand, and in particular extrasystolic arhythmia, are reduced or even disappear under the influence of pulse acceleration.

The polygraphic method records an extreme and permanent arhythmia in these cases. The radial tracing consists of unequal and irregular systoles, constantly varying in duration and power; the jugular tracing generally assumes the so-called "ventricular type," showing a series of oscillations synchronous with the

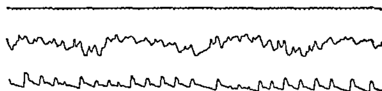


Fig 588—Case 248 H, 56 years; 169 cm., 64 8 kilograms Auricular fibrillation (perpetual arhythmia)

6 23. 1913; pulse, 72 (?); pressures, 135/100 (?), viscosity = 4 3. H = 1200. Albumin absent.

ventricular contractions, but with absence of the *a* wave characteristic of auricular systole. Sometimes, on the most successful tracings, there is noted a series of minute and rapid presystolic undulations, an actual expression of auricular fibrillation (Figs 587 and 588).

Electrocardiography records auricular fibrillation more or less clearly (Fig 586)

series of rapid, irregular oscillations of slight amplitude, *f f f f* (Fig. 586).

Sometimes the fibrillary twitchings of the auricle may even be discerned rather clearly on a good polygraphic tracing (Figs. 587 and 588).

Electrocardiography alone *yields an objective observation* and reliable record of auricular fibrillation.

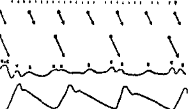
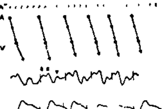
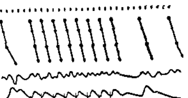
For clinical and practical purposes the diagnosis may, however, be made either with or without the help of graphic procedures.




Fig 586—Electrocardiograms illustrating the 3 leads in a case of mitral stenosis with auricular fibrillation. The *R* wave is very small with Lead *I*, whereas *S* is pronounced, with Lead *III*, *R* appears the highest. There are evidences of hypertrophy of the right ventricle The ventricle is beating very irregularly There is no *P* wave, but on the other hand there are found a number of rapid oscillations, *f*, *f*, resulting from the fibrillation of the auricles (*Cambridge Association*).

The following 3 practical rules may be given in this connection:

1 *Any case of tachy-arhythmia with a heart-rate exceeding 130* is nearly always associated with auricular fibrillation and perpetual arhythmia (the factor of irregularity comprised in the arhythmia excludes instances of increased heart-rate due to fever or emotion, as well as tachycardia of the nervous or paroxysmal types, etc.).

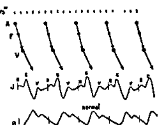
| | | | | |
|---|---|---|---|---|
| | **Paroxysmal tachycardia.** | | | |
|  | Short attack of paroxysmal tachycardia made up of a series of 8 auricular extrasystoles. Each auricular stimulus *is followed* by a corresponding ventricular contraction Abrupt onset and cessation of the attack, with abnormal elongation of the terminal pause | Extrasystolic paroxysmal seizures of a few seconds to a few weeks' duration. Tachycardia exceeding 110 per minute, without fever without exertion, without thyroid enlargement, and uninfluenced by posture. | Do<br>In the long run: Heart failure. | Extrasystolic paroxysms. |
| | ***Sinus (respiratory) arhythmia.*** | | | |
|  | Sinus arhythmia consists of an alternate acceleration and slowing of the *auriculoventricular* cycles of contraction, due to arhythmia of the initial impulse starting from the sinus | A cyclic arhythmia dependent for its rhythm upon the respiration Acceleration during inspiration and slowing during expiration | 0<br>Especially frequent *in* children. | None (undue irritability of the sino-auricular node) |
| | **Auriculoventricular dissociation (heart-block).** | | | |
|  | Complete auriculo-ventricular dissociation Auricles and ventricle contract in a completely independent manner | Paroxysmal, then permanent brady cardia | Syncopal or epileptoid attacks. | Functional auriculo-ventricular dissociation (digitalis, rheumatism, irritation of vagus).<br>*Organic (disease of the* bundle of His) (syphilis, sclerosis, etc). |

# CARDIAC ARHYTHMIAS.

## Normal heart rhythm.

The auricle *A* contracts first and its impulse is transmitted to the ventricle *V* along the bundle *F* The ventricle at once enters into systolic contraction. The time elapsed during transmission, which is practically equal to that occupied in auricular systole, is about one-fifth of a second

R, radial tracing.—J, jugular tracing.
Upper, time tracing Fifths of a second.

| Graphic Representation of the Arhythmias | Kind of Pulse Rhythm | Associated Clinical Features | Physiopathologic Significance |
|---|---|---|---|
| **Extrasystoles (Premature beats).** | | | |
|  The third ventricular systole is premature (ventricular extrasystole) The third auricular systole, occurring during the ventricular refractory period, fails to induce contraction of the ventricle | 'Misstep'' of the heart (intermittences), singly or in groups (bigeminal or trigeminal contractions, etc.). | Functional· Nervousness, dyspepsia, aerophagia. Organic· Myocardial degeneration. | Reflex effect of functional cardiac hyperexcitability (dyspepsia, neurosis). Reflex effect of organic cardiac hyperexcitability (myocardial degeneration). |

Is tricuspid insufficiency always present in perpetual arhythmia, and does recognition of the presence of perpetual arhythmia necessarily mean recognition of the presence of tricuspid insufficiency? Some observers have thought themselves justified in giving a positive answer to this question The author, however, has met with many cases of perpetual arhythmia in which there were no apparent signs permitting of recognition of the presence of tricuspid insufficiency. This latter condition has seemed to him relatively frequent in these cases, but not constant.

Auricular fibrillation and perpetual arhythmia are always associated with and probably dependent upon a deep-seated degeneration of the myocardium and advanced cardiac insufficiency. Their signs are therefore likely to be observed in association with those of myocardial degeneration and heart weakness, *viz*, dyspnea on exertion, cyanosis, venous stasis, passive congestions, hepatic engorgement, edema, reduced output of urine, etc.; and it is very hard to say whether any one of these symptoms is dependent upon them or even whether they are exaggerated through the presence of fibrillation and arhythmia—which, however, is very probably the case.

Attacks of paroxysmal fibrillation with manifest recrudescence of both the arhythmia and the associated symptoms—dyspnea, cyanosis, edema, etc —may be witnessed Other patients, however, seem hardly influenced by the condition. The same is true, indeed, of paroxysmal tachycardia, probably these reactions, seemingly so different, are both actually dependent upon the state of the myocardium, if it is but slightly impaired, the general circulation is comparatively little influenced by recrudescence of the arhythmia; if, on the other hand, it is profoundly degenerated, the usual signs of cardiac insufficiency will rapidly appear.

Mitral stenosis, myocardial degeneration, and arterio-renal sclerosis are the conditions nearly always associated with perpetual arhythmia.

The prognosis of perpetual arhythmia must therefore always be guarded, since auricular fibrillation in itself constitutes a positive sign of more or less advanced degeneration of the myocardium and a probable sign of widespread degeneration

CARDIAC ARHYTHMIAS (*continued*).

| Graphic Representation of the Arhythmias | Kind of Pulse Rhythm | Associated Clinical Features | Physiopathologic Significance |
|---|---|---|---|
| **Alternating pulse.** | | | |
| Alternation of strong and weak contractions | One weak and one strong pulsation succeeding each other at practically equal intervals. | Those of insufficiency of the left ventricle. | Left ventricular insufficiency. |
| **Auricular fibrillation.** | | | |
| The auricular tissue is broken up into many small areas which contract independently. Some of the auricular impulses, at *absolutely irregular* intervals, reach the ventricle, causing it to contract rapidly and irregularly. | Tachyarhythmia exhibiting extreme irregularity and baffling all description (delirium cordis). | Those of cardiac failure. | Auricular fibrillation, especially frequent in mitral stenosis, myocardial degeneration, and arteriorenal sclerosis. |

# ASCITES. [ἀσκός, *a water-bag; an abdomen having the shape of a water-bag.*]

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Ascites, derived from the word ἀσκός, a water-bag, on account of the resemblance of the abdomen distended with serous fluid to such a vessel, consists of an intraperitoneal serous effusion (hydroperitony, dropsy of the peritoneum, dropsy, etc.).

**Recognition of Ascites.**—Accumulation of fluid in the peritoneal cavity is generally a slow, gradual process; in the exceptional cases of ascites spoken of as *a frigore*, due to sudden obstruction of the portal vein, effusion may take place rapidly.

Though sometimes obvious, especially when it has attained a certain size, when the abdominal parietes are relatively thin, and when the fluid is freely movable, its recognition may in other instances be attended with considerable difficulty when, as is frequently the case, the parietes are thick and infiltrated, when the effusion is of slight or moderate extent, when its mobility is limited owing to adhesions, etc.

Clinically, its recognition is to be based on a systematic examination of the case:

**Inspection.**

(*a*) **Shape of the Abdomen.**—1. *Vertical position:* Abnormal prominence of the hypogastrium and the iliac fossæ.

2. *Recumbent position:* The flanks broaden and flatten out, like the abdomen of an amphibian, the fluid moves about, on the whole, according to gravity, therefore passing to the dependent side when the patient is in lateral decubitus

3 Occasionally the *umbilicus, turned out like a glove finger,* forms a small, soft, fluctuating, depressible, translucent tumor.

(*b*) **Condition of the Skin**—The skin is frequently smooth, white, even, polished, and shining; sometimes thickened, infiltrated, and edematous; at times erythematous Striæ comparable to those of pregnancy may be noticed upon it

Nevertheless—and in this condition the therapeutic test is often conclusive—certain instances of perpetual arhythmia are markedly reduced by well-directed drug treatment, while others are completely refractory. The prognosis is obviously profoundly influenced by these factors. On the whole, the same conclusion always follows, *viz.*, that the prognosis is governed much more by a study of the contractility of the heart muscle than by a study of its conductivity.

movements of the abdominal parietes, which would constitute a source of error.

At all events, fluctuation is almost pathognomonic of ascites, as it occurs in the absence of ascites only in a few exceptional cases of ovarian cyst with thin walls

**Vaginal Palpation.**—With this procedure, a doughy, firm condition in the culs-de-sac is occasionally found.

Sometimes vaginal palpation may prove of service in affording an early diagnosis of ascites (descent and reduced weight of the uterus, and extreme mobility of the cervix).

**Functional Evidences.**

**(a) Chiefly manifestations due to distention and pressure.**

1. *Increased size of the abdomen* · The patient can no longer button up his trousers.

2. *Digestive disturbances:* Constipation due to pressure on the intestines, resulting in paralysis (Chopart's law: Any muscle underlying an inflamed serous membrane is paralyzed) Indigestion and tympanites

3. *Urinary disturbances;* Dysuria, diminished secretion of urine, oliguria, and opsiuria, chiefly on account of the loss of fluid resulting from the ascitic accumulation

4. *Cardiopulmonary dyspnea* due to pressure on the diaphragm, causing reduced lung expansion and displacement of the heart.

5. *Edema of the lower extremities,* due either to the same cause as the ascites or to pressure exerted on the inferior venæ cavæ.

**(b) Functional disturbances attending the primary disorder** (cirrhosis, peritonitis, etc ).

**With What Conditions Might Ascites be Confounded?**—(*a*) **Abdominal Meteorism.**—Here, resonance and tympany are increased; yet, in truth, ascites is frequently accompanied by meteorism; in fact, meteorism may conceal an ascites. The best differential sign in these cases, in the author's estimation, is the displacement of the dullness upon assumption of lateral decubitus, the dullness appearing in the most dependent area, at a point previously resonant.

(*b*) **Edema of the Abdominal Wall.**—The finger leaves a depression in the parietal tissues; dullness is uniformly distrib-

(*c*) **Superficial Venous Circulation.**—The accessory portal veins, normally only slightly developed, enlarge, sometimes to a considerable size, in the event of obstruction to the blood-current through the portal vein. The circulation is in part re-established through these vessels, whence there occurs dilatation of the subcutaneous veins of the abdomen, with the production of a prominent network of veins between the pubis and the xiphoid appendix, especially on the right side, in the form of a peri-umbilical venous plexus.

(*d*) Sometimes there is **concomitant hydrocele,** owing to persistence of the vagino-peritoneal duct.

**Palpation.**—The abdomen exhibits an even, tense, sometimes elastic, and firm enlargement.

The fluid present masks the intestinal mass and forms an obstacle to detailed examination of the abdominal viscera, whence the necessity of puncture in cases where such examination is imperative.

**Percussion.**—Flatness manifestly varies in extent according to the amount of effused fluid.

1. Its primary localization is over the iliac fossæ and the hypogastrium (fluid); the umbilical and epigastric regions are generally the seat of tympanitic resonance (intestines), the transition from flatness to tympany being, however, gradual.

2. A small effusion may be unrecognizable on percussion; but if the patient is turned on the side, the fluid will collect on that side and flatness be detectable.

3. The flatness exhibits movable margins, which vary according to the patient's position if the ascites is movable and free; it is fixed, however, if the effusion is encysted or walled off (Fig 596).

**Combined Palpation and Percussion.**—This procedure yields one of the most important indications of ascites, *viz., fluctuation*

With one hand applied flat over one side of the abdomen, the physician taps lightly with the other on the opposite side, either by light percussion or by flipping a finger; the former hand notes a sensation as of a blow or wave. It is often useful to have some one else apply the ulnar border of one hand along the linea alba, in order to prevent transmission of wave-like

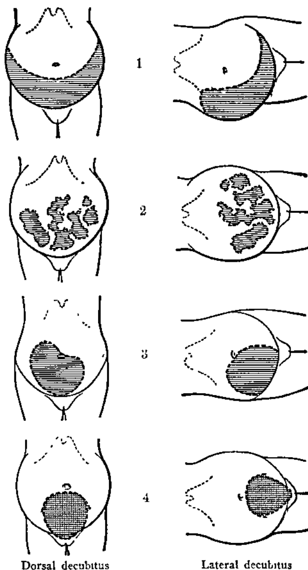



Fig. 596—Abdominal areas of flatness in ascites and various other abdominal disorders

1. Frank ascites of hepatic origin 2 Encysted ascites of the peritoneal type. 3 Ovarian cyst 4 Pregnancy, retention of urine.

uted, and fluctuation is absent. Abdominal edema and ascites, however, frequently coexist.

(*c*) **Retention of Urine.**—The bulging and dullness are distinctly located in the hypogastrium, with their convex aspect directed upward—the opposite of the condition in ascites. Urine is passed by overflow; palpation of the hypogastrium is more or less painful.

In the event of doubt, which is seldom excusable, catheterization of the bladder will dispel both the doubt and the abdominal enlargement

(*d*) **Pregnancy.**—The enlargement is hard, in the median *line, rounded, pyriform, and hypogastric in situation. Palpation* reveals that the uterus is the cause of the bulging. The convex aspect is directed upward—the opposite of the condition in ascites. On the whole, the mere thought of the possibility of pregnancy is enough to eliminate this source of error. If doubt should nevertheless exist, the other signs of pregnancy should be examined for, *viz*, cessation of menstruation, secretion of colostrum and other mammary changes, progressive enlargement, and after four and a half months, the diagnostic fetal signs.

(*e*) **Ovarian Cysts.**—The following distinguishing features may with advantage be recalled:

1. *Shape of the abdomen.*—Globular, and with umbilicus normal, in ovarian cyst

Flattened out and with prominent umbilicus in ascites.

2. *Flatness* —In ascites · Flatness in the lumbar regions; umbilical *region resonant;* flanks flat; the flat area is, in a general way, convex below; the areas of flatness are movable and vary with the position of the patient.

In ovarian cyst Resonance in the lumbar, iliac, and epigastric regions; flatness rather median and hypogastric in location, and sometimes umbilical, with convexity directed upward; flatness is not changed by altered position of the patient.

3 *Fluctuation.*—Practically constant in ascites; exceptional in ovarian cyst.

4. *History of case.*—Often negative in ovarian cyst.

type (*e.g.*, alcoholism with tuberculosis, alcoholism with syphilis, syphilis with tuberculosis, or even alcoholism with tuberculosis and syphilis).

(*b*) **Ascites of Peritoneal Origin (General Type: Tuberculous Peritonitis).**—1. The ascites is of moderate extent, often but slightly fluctuating, and advancing by alternate exacerbations and recessions

The fluid is but slightly movable, and frequently encysted.

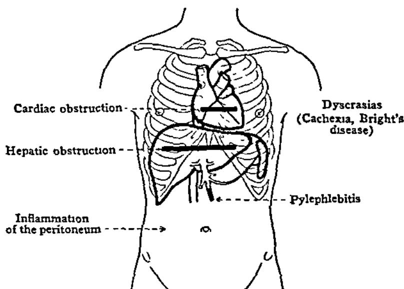



Fig 597—The causes of ascites
Cardiac—Hepatic—Peritoneal.—Pylephlebitic.—Dyscrasic.

Sometimes there are coexisting dull and hard infiltrations, best palpated after puncture.

2. The concomitant signs should be looked for, *viz*, peritoneal and pleural friction rubs, pleural effusion, ganglia, signs of pleuropulmonary, genital, or articular tuberculous disease or typhobacillosis, fever, vomiting, etc

3 The ascitic fluid is serofibrinous, containing a much larger amount of fibrin, protein, or cells than that of mechanically produced ascites In short, it presents the typical features of inflammatory exudates Guinea-pig inoculation will give positive results in the presence of tuberculosis Certain laboratory

Always positive in ascites, comprising such features as hepatic disorder (cirrhosis), cardiac affections, general impairment of health (tuberculous peritonitis), some serious organic affection (cachexia), etc.

Notwithstanding all these distinguishing features, many mistakes are made, the possibility of coexisting ascites and cyst further complicates matters.

**Causes of Ascites.**—In the presence of ascites, attention should be particularly directed to the *liver, peritoneum,* and *heart.*

The diagnosis is based largely on the history of the case, the existing physical signs, the course of the ascites, and the nature of the fluid withdrawn by puncture.

*(a) Ascites of Hepatic Origin (General Type: Atrophic Cirrhosis; Typical Ascites).*—The onset is slow, gradual, more rarely abrupt, rapid after exposure to cold; the fluid is free in the abdomen, movable, and fluctuation is readily elicited.

The history includes a more or less well-defined precirrhotic stage, characterized by manifestations of increased portal pressure (hemorrhoids, collateral circulation), indigestion and gastro-intestinal disturbances, catarrhal disorders, meteorism, diarrhea, hepatic congestion and incipient jaundice, reduced output of urine, etc.

1. The course of the disease is progressive.

2. The liver is always found diseased, generally reduced in size (Laennec's atrophic cirrhosis), sometimes enlarged (hypertrophic or alcoholic cirrhosis of Hanot and Gilbert); the spleen shows enlargement; there are manifest evidences of portal hypertension, together with distinct impairment of nutrition. Ascites may be encountered in syphilis of the liver or in primary or secondary nodular cancer.

3. The fluid obtained by puncture is serous and poor in fibrin, cellular elements, and protein material.

4. Nevertheless, it is well to bear in mind that peritonitis may and frequently does accompany many forms of cirrhosis, and that cases of fibrous, alcoholic, tuberculous, and syphilitic hepatitis are quite often associated with localized peritonitis (perihepatitis) or generalized peritonitis of a similar or hybrid

est forms, such as the long standing idiopathic ascites of young girls, running a slow course, without fever or constitutional disturbances, and nearly always ending in spontaneous recovery (dropsy of the peritoneum), to the most serious varieties, such as the ulcero-caseous forms which lead so quickly and inevitably to the hectic state.

Finally, reference should be made to the frequent, not to say constant, participation of the pleura in the process of peritoneal tuberculous involvement, almost always constituting more properly a *pleuro-peritoneal tuberculosis* At all events and for practical purposes, in the presence of a suspicious peritoneal disorder the physician should systematically examine the pleuræ and *always* perform an exploratory puncture of the pleura; this procedure will supply a solution of the problem in the majority of cases

**(c) Ascites of Cardiac Origin (General Type: Heart Failure).**

1. One of the most specific features of these cases is that here the ascites clearly follows other manifestations of edema, as in the lower extremities, scrotum, and lumbar regions—in contrast with what occurs in cirrhotic and peritonitic ascites,—and that it is frequently combined with hydrothorax.

2. The diagnosis is clear from observation of the attending circumstances, the coexisting heart failure, and the physical signs of cardiac disease At the most, doubt might occur in the very advanced cases in which—the primary hepatic cirrhosis having led to secondary cardiac insufficiency, or, on the other hand, primary heart disease having resulted in cirrhosis of cardiac origin—a combined cardio-hepatic inadequacy, manifested in both heart failure and hepatic cirrhosis, has been produced A careful inquiry into the past medical history as regards the heart and liver, along with observation of the course of the edema and the heart sounds, will nearly always settle the question of priority, even though such an aim is of academic rather than practical interest, since the therapeutic indications, like the disturbances of function, are in close combination in these cases

**Other Causes**—Apart from the above three cardinal sources of ascites, mention should be made of:

procedures are available for examining ascitic fluid for the tubercle bacillus (Jousset's *inoscopy*).

4. It is well to note that tuberculosis of the peritoneum comprises by far the greatest number of the cases of *peritonitic ascites*, but that one should also think of the possibility of *cancer of the*

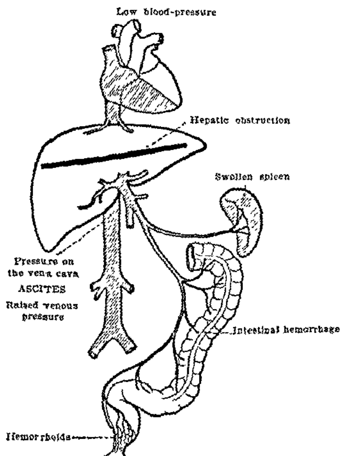



*Fig 598—The syndrome of increased portal pressure*

*peritoneum* in an old, cachectic individual. The reactions involving the lymph-glands; examination of the fluid, which generally contains red blood cells; the history of the case, and the subsequent course of the disease, will soon confirm the latter diagnosis if the possibility of the condition is merely recollected.

Nor should it be forgotten that *tuberculous peritonitis with ascitic effusion* may occur in any grade of severity, from the mild-

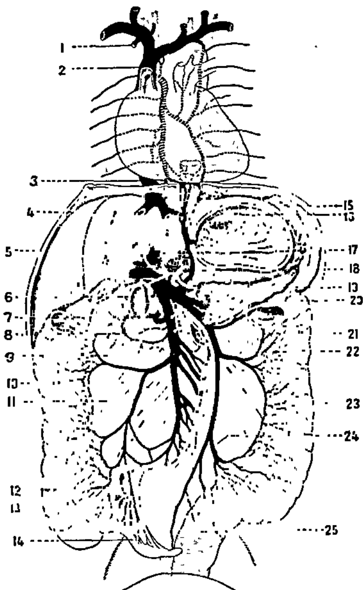



Fig 600—Tributaries of the portal vein (after *Bérard* and *Vignard*). *1* Internal mammary vein *2* Superior vena cava *3* Inferior vena cava. *4* Suprahepatic vein. *5* Liver. *6* Trunk of the portal vein *7*. Gastro-duodenal veins *8* Pancreas *9* Duodenum *10*. Mesentery *11* Ileocolic vein *12*. Ileocecal angle *13* Appendicular veins *14* Appendix. *15* Spleen *16*. Stom
vein *18* Celiac axi
*21*. Inferior mesenter
Colosigmoid vein *2*

1. **The Ascites of Nephritis.**—This is, on the whole, rather uncommon and is associated with the usual signs of Bright's disease (see *Albuminuria* and *Edema*). The high urea content in the ascitic fluid of azotemic cases should be borne in mind.

This diagnosis should not be accepted unless one has become convinced from careful examination of the absence of hepatic cirrhosis, peritoneal inflammation, or heart weakness.

The differential features include, more particularly: Albu-*minuria and, in the absence of all cardiac inadequacy, precession* of the edematous manifestations (lids, extremities, and scrotum).

2. **The Ascites of Cachexia.**—This is likewise an exceptional

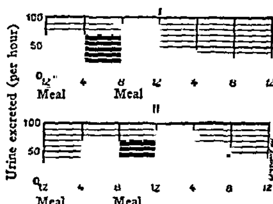



*Fig 599—Normal rhythm of urinary elimination (above). Opsiuria (below)*

form, and one which, upon thorough investigation, can nearly always be relegated to one of the three above-mentioned groups, *viz.*, hepatic, peritoneal (tuberculous or neoplastic), or cardiac ascites.

3. **Chylous Ascites.**—Also a rare form, at least in our own countries, and doubtless of varied and complex causation.

*The fluid withdrawn is whitish, opalescent, milky in appear-*ance, containing little protein but much fat, which is dissolved by ether, thereby clearing up the fluid Its composition is somewhat similar to that of pus (hyperleucocytosis).

In the presence of this form of ascites there have been detected filariasis (Lancereaux), tuberculosis (Courtois-Suffit), *chronic inflammation of the peritoneum (Letulle), and pressure* on the thoracic duct by enlarged mediastinal glands (Strauss).

| CAUSES. | HISTORY. | FEATURES OF THE ASCITES | CONCOMITANT CLINICAL SIGNS. | PROGNOSIS. |
|---|---|---|---|---|
| Of nephritic origin | That of Bright's disease | Those of ascites of hepatic origin. | Albuminuria. *Chloridemia* High blood-pressure Precession of edema. | That of the primary nephritis. |
| Of dyscrasic (cachectic) origin | That of cachexia (tuberculosis, syphilis, cancer, arteriosclerosis, etc ) | Variable, according to the cause | Those of the primary disorder (tuberculosis, syphilis, cancer, etc.) | Fatal. |
| Of pylephlebitic origin | Abdominal infection; peritonitis, appendicitis | *Sudden onset*<br>Early recurrence after puncture. | Pain, diarrhea, vomiting, hematemesis and intestinal hemorrhage<br>Marked splenic enlargement.<br>Tremendous collateral circulation. | Generally grave. |
| Chylous ascites, | Filariasis.<br>Tuberculosis<br>*Chronic peritoneal* inflammation. | Chylous, milky, opalescent fluid, containing many *leucocytes.* | Those of the causal disorder. | Variable according to nature of case. |

| Causes | History. | Features of the Ascites | Concomitant Clinical Signs | Prognosis |
|---|---|---|---|---|
| Of hepatic origin.<br>Type case: Atrophic cirrhosis. | Digestive precirrhotic disturbances<br>Portal hypertension<br>Digestive disorders<br>Hepatic congestion<br>Frequently alcoholic excesses | **Typical ascites, freely movable, and with marked fluctuation**<br>Serous fluid containing but little fibrin, protein, or cells. | **Changes in the liver:** Atrophy. Alcoholic atrophic cirrhosis.<br>*Hypertrophy* · *Alcoholic* hypertrophic cirrhosis.<br>Irregular Nodular cancer<br>Lobulated Syphilis of liver<br>**Enlarged spleen: Portal hypertension.** | Hypertrophic cirrhosis: Favorable<br>Atrophic cirrhosis: Grave<br>Syphilitic cirrhosis: Serious<br>Malignancy. Fatal. |
| Of peritoneal origin<br>Type case: Tuberculous peritonitis. | Congenital debility<br>Impaired general health<br>Evidences of tuberculous disease. | **Ascites but slightly movable,** confined by tissue overgrowth and adhesions<br>Serofibrinous, inflammatory fluid, containing much fibrin, protein, and cells. | Pleural effusion almost constant.<br>Signs of concomitant tuberculous infection<br>Positive guinea-pig inoculation. | Ranges from the mildest ("idiopathic" ascites of young girls) to the most serious (ulcerocaseous forms) (cancer, exceptional, to be thought of). |
| Of cardiac origin.<br>Type case: Heart failure. | **Cardiac** disturbances.<br>Inadequacy or failure of the heart | Those of mechanical ascites of *hepatic origin.* | Heart lesion.<br>**Manifest evidences of cardiac insufficiency.**<br>Precession of edematous manifestations. | That of the primary heart disorder. |

# ASTHENIA AND FATIGUE. [*from α, privative; σθένος, strength. Deprived of strength.*]

The term fatigue relates to a sensation too commonly experienced and well characterized to require definition. At most need it be said that all grades of fatigue may be encountered, from the mild, temporary, almost pleasant sensation of fatigue, manifested in a desire to rest, to deep, persistent, and lasting exhaustion, almost completely depriving the individual of will power and of the ability to act Lastly, it is necessary to make a distinction between "paralysis," or abolition of voluntary movements of some portion of the body, and "fatigue," which merely renders motion distressing or actually painful; as a matter of fact, however, "paresis" is in some respects closely allied to "fatigue."

Fatigue as a symptom is too common and ordinary a manifestation of most infectious, toxic, or depressive states to lend interest even to an attempt at a complete semeiologic description in this connection The scope of this chapter will therefore be restricted

1. To recalling the commoner clinical states in which fatigue occurs as a symptom

2. To recalling under what circumstances fatigue, by virtue of its unusual persistency, intensity, or variety, assumes definite clinical significance and is sometimes practically pathognomonic.

The feeling of fatigue may be physiological, *i e.*, normal, after prolonged physical or mental exertion, after some form of shock to the system, violent emotion, or prolonged test. In this instance it is merely accidental and temporary. It yields readily to rest, sleep, and removal of the cause.

Fatigue may assume an abnormal, pathologic type by virtue of:

Its intensity (exhaustion, profound asthenia).

4. **The Ascites of Pylephlebitis.**—An altogether exceptional form characterized by its sudden onset, its exceedingly prompt recurrence after puncture, the attendant pain, diarrhea, vomiting, and hemorrhage in the digestive tract, the splenic enlargement, and the pronounced development of collateral circulation in the abdominal wall.

| Causes | Blood-pressure | Sergent's White Line | Blood Examination | Urine Examination | Temperature | Associated Clinical Signs |
|---|---|---|---|---|---|---|
| **I.—Nervous.** | | | | | | |
| Organic diseases of the nervous system | ? | ? | Variable | ? | 0 | Those of the pathologic condition: Hemiplegia, etc. |
| Depressive psychoneuroses (psychasthenia, neurasthenia, etc.) | — | ? | ? | ? | 0 | General depression, aboulia, anxiety, etc. |
| **II.—Humoral.** | | | | | | |
| (a) Anemias. | — | ? | Reduction of red cells | 0 | 0 | Pallor, including mucous membranes |
| (b) Hyposphyxia. | — | ? | Hyperviscosity | 0 | 0 | Livid aspect, cryesthesia, dyspnea on exertion |
| (c) Glandular insufficiencies. Adrenal insufficiency. | — | + | Hyperviscosity. | Sometimes temporary glycosuria | 0 | Bronzing in Addison's disease. |
| Myxedema. | — | ? | ? | 0 | 0 | Doughy infiltration of tissues. Urinary insufficiency |
| (d) Diathetic disorders. Obesity | + or — | 0 | ? | 0 | 0 | Overweight. |
| Diabetes | + | 0 | Hyperviscosity | Glycosuria. | 0 | Polyuria, polyphagia, polydipsia |
| (e) Autotoxic states. Uremia | + | 0 | Hyperazotemia | Albuminuria | 0 | Headache, general torpor, digestive disturbances |
| Arteriosclerosis | + | 0 | Hypoviscosity. Hyperazotemia | Albuminuria | 0 | Vertigo, epistaxis, cardiac hypertrophy |
| **III.—Infectious.** | | | | | | |
| (a) Acute (typhoid, influenza, malaria, scarlet fever, diphtheria, etc. | — | + or 0 | Hyperviscosity | Evanescent albuminuria. | + | Symptoms of infection. |
| (b) Chronic: Tuberculosis. | — | + or 0 | Hyperviscosity. | 0 | + | Impaired general health. Auscultatory and x-ray evidences. |
| Syphilis. | — | ? | Hyperviscosity. Wassermann + | 0 | + or 0 | Manifestations in the skin, mucous membranes, or viscera — Wassermann +. |
| Malaria. | — | + | Plasmodium during paroxysms. | 0 | + | Intermittent fever. Enlarged spleen |

Its duration (yielding neither to rest nor to removal of the cause).

Its special modalities, being frequently periodic in type.

Its location, often in the lumbar regions.

It may be succinctly recalled that *the more usual causes of* pathologic fatigue *may be*

I. **Nervous.**—Nervous fatigue results naturally from overwork, repeated emotion, and in particular from insomnia of whatever cause

It is met with almost constantly, either as a subsidiary or principal manifestation, in the majority of *organic diseases of the nervous system, and in particular in all paralytic states, to which no further reference will be made.*

Its diagnostic significance may be very great in all forms of depressive psychoneuroses, *viz*, neurasthenic and neurastheniform states, anxiety neurosis, cerebrocardiac neuropathy, psychasthenia, general asthenia with gastrointestinal atony, ptosis, and impaired nutrition, sympatheticotonia, etc. It is constant in all these conditions and sometimes predominant and overmastering; it is nearly always associated with insomnia. The cause will always be found to be overstrain, physical or mental; sexual excesses, or some emotional shock, the patient himself not generally connecting, however, the two groups of phenomena. Once this cycle has been established, *viz.*, excessive excitability ("emotionalism," suggestibility) and asthenia (insomnia), it tends, as an actual vicious circle, to persist, the "emotionalism" and suggestibility engendering or accentuating the asthenia and insomnia, and the asthenia and insomnia, in turn, engendering or accentuating the "emotionalism" and suggestibility.

The diagnosis is generally easy, an inquiry into the patient's mental state yielding conclusive results It is to be borne in mind, however, that a diagnosis of primary psychoneurosis should never be made except after a process of exclusion, and that the practitioner should always make certain that it is not symptomatic of some somatic disorder such as tuberculosis, arteriosclerosis, syphilis, azotemia, etc.

however, at times lead to the detection of an incipient diabetes in a previously healthy subject; acetonemia or azotemia will do the same in a subject with glycosuria of long standing (see *Glycosuria*).

(*e*) **Autotoxic States.**

1. Uremia (and especially azotemia).—A *persistent, overpowering asthenia* in conjunction with general *torpor* occurs almost constantly in *azotemia.*

2. Arteriosclerosis (senile degeneration).—The same statement applies to arteriosclerosis.

III. The **infectious causes** of "fatigue" and asthenia manifestly induce them through poisoning of the nervous and muscular tissues by toxins and through adrenal insufficiency. Strictly speaking, they should therefore be included in the groups already given. Let it be again repeated that in the classification herein adopted "dogmatic logic" is deliberately sacrificed in the interests of "practical pragmatism"

The infectious forms of fatigue are often plainly evident; this applies to most of the asthenic conditions witnessed in conjunction with acute infections, *e g*, typhoid and post-typhoid asthenia, influenzal and post-influenzal asthenia, diphtheritic and post-diphtheritic asthenia, etc In any case careful inquiry should be made to determine (1) whether this post-infectious asthenia is not concealing incipient tuberculous disease, and (2) whether it is accompanied by pronounced signs of adrenal insufficiency (low blood-pressure, asthenia, Sergent's white line)

Particular attention should be paid to the **chronic, sluggish, cryptogenic forms of asthenia.** In this connection especially should the physician refuse to be satisfied with such "easy" diagnoses as "anemia" or "neurasthenia," but should, on the contrary, make a deliberate search for the three great chronic infections (**tuberculosis, syphilis,** and *malaria*) and the three main varieties of intoxication already referred to (**uremia, glycosuria,** and *hyposphyxia*).

No detailed reference need be made to the well-known signs of these disorders Yet it may be recalled in conclusion that any case of persistent, unaccountable asthenia should lead one to the particular thought of the possibility of incipient tubercu-

**II. The humoral causes** of fatigue are legion. *Any form of intoxication,* whether endogenous or exogenous and toxic or toxinic, may cause asthenia. However defective the following classification may be—and it must be confessed that many of the groups overlap—it may be adopted, even if merely for mnemotechnic purposes.

(*a*) **Anemias.**—In this group fatigue is continuous and is associated with the customary indications of anemia, *viz*, pallor of the mucous membranes, reduction of red cells, reduction of hemoglobin, etc., together with anorexia, indigestion, etc. The main object, however, should be, trace the cause of the anemia (see *Anemias*).

(*b*) **Hyposphyxic States.**—These involve a special circulatory syndrome characterized essentially by low blood-pressure and relatively high blood viscosity, these causing all the usual manifestations of impaired circulation, *viz*, cyanosis, lowered superficial temperature, dyspnea, and undue fatigability (see *Low blood-pressure*).

(*c*) **Conditions of Glandular Insufficiency.**—Particular attention will here be paid to *Addison's disease* and *myxedema.*

1. Addison's disease (adrenal insufficiency).—*Addison's disease* constitutes simply a very serious, but exceptional, form of *adrenal insufficiency,* which recent observations, particularly those of Sergent, have shown to be so common in all infectious and post-infectious states (typhoid fever, scarlet fever, dysentery, malaria, cholera, tuberculosis, etc ). During the course of these disorders the 3 cardinal signs should be systematically looked for: *Asthenia, low blood-pressure, and Sergent's white line.*

2. Myxedema.—The characteristic doughy infiltration of the tissues (myxedema), the repeated intermissions in the progress of the disease, and the permanent asthenia lead to the diagnosis.

(*d*) **Diathetic States** (Metabolic Disorders).

1. Obesity.—This in itself is often dependent upon insufficiency of several of the ductless glands, and particularly of the thyroid, which allies it in some respects to myxedema *Asthenia* should lead to a suspicion of the latter condition (see *Obesity*).

2. Diabetes.—Ordinarily the diabetic is a supernormal, overactive, indefatigable individual. *Asthenia* of unknown origin may,

# CHILLS.

[*French* Frissons, *from* frigere, *to be cold.*]

A chill consists essentially of a sudden tremor of varying extent and varying duration, usually accompanied by a sensation of cold and followed by a sensation of warmth

At least three grades of chills may be recognized

*Cryesthesia or chilliness.* An unpleasant sensation of cold, with very slight tremor.

*Shivering*

An actual *major chill*, involving the entire body and attended with chattering of the teeth, diffuse and violent trembling, and an intense feeling of cold.

All true chills, except the nervous or emotional chills, are followed by fever As a general rule, a definite chill accompanied by an abrupt rise of temperature is symptomatic of the onset of an infectious disease, by far the most frequent of such disorders being pneumonia, grippe, malaria, tonsillitis, and septicemia.

The commonest causes of chills may be enumerated as follows:

- **Pyogenic and septicemic infections:**
  - *Pneumonia, tuberculosis, appendicitis*
  - *Septic wounds*
  - *Suppurative disorders of the liver and kidneys; biliary and urinary infections.*
  - *Tonsillitis.*
  - *Vegetative endocarditis.*
  - *Phlebitis*
  - *Empyema.*
  - *Erysipelas.*
- **Malaria.**
- **Renal and hepatic colic.**

Nothing special need be said concerning:

losis, evidences of which should be sought with great care, *viz.*, (1) *Functional:* weakness, lassitude, dyspnea on exertion, anorexia, loss of weight, slight vesperal fever, night sweats, cough, frequent heart rate, and hemoptysis. (2) *Physical:* reduced breathing capacity, slight impairment of resonance at one apex, increased vocal resonance, and persistent abnormalities of respiration at one apex (rough, jerky breathing; rough, prolonged, and blowing expiration, friction rubs, etc.).

Fluoroscopy, while of very great value, has in no wise detracted from the significance of the above time-honored physical signs.

Systematic clinical examination will automatically yield a solution to these problems, provided it is, or attempts to be, thorough and comprehensive

**Systematic Clinical Examination of Asthenic Subjects.**

| | |
|---|---|
| 1 Determination of blood-pressure, systolic and diastolic. | |
| High blood-pressure | Arteriosclerosis, nephritis, azotemia. |
| Low blood-pressure | Hyposphyxia, tuberculosis, adrenal insufficiency. |
| 2 Blood examination. | |
| Low red cell count. | Anemia |
| Hyperviscosity | Hyposphyxia, azotemia, tuberculosis, adrenal insufficiency, acetonemia |
| Hyperazotemia. | Azotemia |
| Wassermann reaction | Positive Syphilis |
| 3 Temperature. | |
| Hyperthermia. | Infectious states (tuberculosis, malaria, etc.) |
| 4 Auscultation. | |
| Lungs. | Tuberculosis |
| Heart | (Accentuation of second sound, gallop rhythm: Nephritis, arteriosclerosis). |
| 5 Urine examination. | |
| Sugar, acetone | Diabetes, acetonemia. |
| Albumin. | Albuminuria (azotemia) |
| 6 Examination of reflexes and nervous reactions. | 1. Psychoneuroses<br>2. Sergent's white line· Adrenal insufficiency. |

# COMA.

[Κῶμα. *Drowsiness, suspension of the mental functions.*]

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Coma consists of a state of profound somnolence with more or less complete loss of consciousness, sensibility, and motility. While it is the most striking feature of the syndrome resulting from apoplexy, it may be and frequently is met with under other circumstances

It could hardly be confused with the deep sleep of convalescents or hysterical cases, or with syncope or asphyxia.

The deep sleep of convalescents is a quiet sleep, with the pulse regular and respiration normal. It is seldom so profound that the patient cannot be awakened by some sharp stimulus, and the history of the case will generally exclude the idea of coma.

The sleep of hysterical patients might more readily lead to confusion Only rarely, however, will the previous history and the features of the onset of the attack fail to point the way to a proper diagnosis, which will be thoroughly illuminated, furthermore, by systematic investigation of the hysterogenous zones.

Systematic diagnosis of hysterical pseudo-coma may be said to be based on the following clinical findings:

1. If the onset was sudden, the patient falling, this fall occurred without the patient receiving any severe blow or any wound or traumatism; no biting of the tongue, and no relaxation of the sphincters

2 Frequently there are noted contracture, winking of the lids, and various movements of the eyeballs, which are absent in true coma.

3 The patient, while apparently insensitive to pain, noise, and light, reacts to an exaggerated degree, on the other hand, to pressure upon a hysterogenous zone, to a cold affusion, or to appropriate suggestion in a loud voice. The author has wit-

The *chill from exposure* or *"a frigore."*

The *emotional chill,* or shivering "with fright" or "with horror," or simply the psychoneuropathic chill. Some nervous degenerates, apparently "constitutional shiverers," are always ready to shiver on the slightest provocation

| CAUSES. | FEVER. | BLOOD EXAMINATION | CLINICAL SIGNS | MEANS OF RECOVERY. |
|---|---|---|---|---|
| Nervousness. | None | Negative. | Neuropathic stigmata. | Suggestion. Discipline |
| Septic states. | Remittent. | Polymorphonuclear leucocytosis. | Local or deep-seated visceral infection. Infectious endocarditis. | Dressings, operation, and drainage. Collargol injections. |
| Phthisis. | Remittent. | Frequently leucocytosis. | Stethoscopic and fluoroscopic pulmonary evidences | General and local treatment. Hygienic measures. |
| | | | Tubercle bacilli in sputum. | Spontaneous recovery. |
| Pneumonia. | Continued. | Frequently leucocytosis | Stethoscopic evidences. Characteristic sputum. | Hygienic treatment. General measures Spontaneous recovery. |
| Hepatic colic. | Remittent or intermittent. | Not characteristic. | Hepatic colic or gastralgia Frequently jaundice. Pain in right hypochondrium. | Morphine. Operation. Diet. |
| Malaria. | Intermittent (attacks at definite intervals) | Leucopenia Malarial parasites. | Splenic enlargement | Quinine. Arsenic |
| *Typhoid fever.* | *Continued* | [illegible] ture | [illegible] | [illegible] treat- |

The history *is of prime importance.*

1. Has there been some traumatic injury, fall, or contusion preceding the coma? If so, fracture of the skull.

2. Does the patient give a history of similar seizures before, and is he subject to convulsive attacks? Epilepsy, eclampsia, uremia.

3 Has the patient been intemperate? Had he been on an alcoholic spree before the coma came on? Alcoholism

4. Has the patient had syphilis? Has he previously had treatment for this disease? Was he under treatment at the time? Brain syphilis

5 Did the patient have nycturia, vertigo, albuminuria, etc.? Interstitial nephritis, arteriosclerosis, uremia, etc

6. Was the patient in a rundown state, did he pass very much water, had he had itching and digestive disturbances for a few days, etc? Diabetes

And so on

In short, the practitioner should carefully take note of all information supplied by the relatives as to the patient's previous medical history, such information will often yield highly serviceable clinical indications

*The* direct clinical examination *is of much greater importance still.*

As in other conditions, it should be conducted systematically and comprehensively. The following lines of inquiry are, however, particularly essential

1 **Is hemiplegia present?** (See *Hemiplegia*). With decrease of muscle tone on one side of the body, sometimes conjugate deviation of the head and eyes, an exaggerated patellar reflex on one side, and the Babinski sign or plantar reflex Hemiplegia, if present, will usually constitute a clinical expression of brain hemorrhage or softening, it may be met with in uremia, and in some forms of meningitis in childhood.

2. **Is fever present?** And if so, was it present before the coma (typhoid fever, cerebral rheumatism, tuberculous meningitis, cerebrospinal meningitis)? Or was it present along with the coma (pernicious malarial fever)? Or did it come on after the coma (certain forms of cerebral hemorrhage)?

nessed many seizures of this type, sufficiently striking to alarm an experienced hospital staff, suddenly cease upon suggestion that the patient was to be "immediately placed in isolation in the special quarters" or to be "subjected to cauterization with the hot iron." Much oftener, indeed, it yielded to brief occlusion of the nose and mouth.

4. Lastly, these hysterical pseudo-comatose states are frequently associated with postures apparently unconsciously assumed, but plainly semi-voluntary to the close observer; the same applies to the pseudo-delirium encountered in these subjects.

**Syncope** is ordinarily of short duration The sudden loss of consciousness, pallor, weakness, and even almost complete arrest of the heart-beats, the rapidly beneficial effect of the horizontal posture, elevation of the legs, and stimulating injections, will preclude any prolonged hesitation.

In *asphyxia*, the history of the case, the cyanosis, the livid appearance, and the reduced temperature of the lower extremities will obviate any mistake.

**The ordinary causes of coma** may, for practical purposes, be enumerated as follows

**Toxic causes:** Exogenous Alcohol, opium
Endogenous· Uremia, acetonemia, acidosis (diabetes).

**Cerebral causes:** Vascular Apoplexy, hemorrhage, thrombosis, or epilepsy.
Inflammatory: *Meningoencephalitis.*
Neoplastic· Brain tumors.
Traumatic: Skull fractures.

**Infectious causes:** Malaria, rheumatism, typhoid fever, or infectious jaundice

**Circulatory causes:** Stokes-Adams' disease.

Coma having been clinically encountered, the causal diagnosis, which is of prime importance both because of the prognosis it affords and the indications it gives for treatment, is based chiefly upon the *history* of the illness and, in particular, on the results of *clinical examination.* The history and clinical findings must be systematically collated.

| | History | Hemi-plegia | Fever | Urine | Blood | Blood-pressure | Kernig's Sign | Cerebrospinal Fluid |
|---|---|---|---|---|---|---|---|---|
| **Post-traumatic.** | Trauma. | Occasional | — | — | — | — | Constant. | — |
| **Inflammatory (meningo-encephalitis).** | Headache. *More or less* rapid onset. | Exceptional<br>Contractures | Constant, irregular | Frequently albuminuria | Leucocytosis. | | | Pathogenic bacteria. Differential white count more or less characteristic. |
| **Toxic (opium)** | Habitual, occasional, or accidental intoxication | —<br>Pinpoint pupils. | — | — | — | — | — | — |
| **Neoplastic (brain tumors).** | Headache<br>Dizziness<br>Vomiting<br>Disturbances of vision. | Frequent. | Exceptional | Frequently albuminuria | — | Frequently high blood-pressure. | Rare | High blood-pressure. |
| **Demential (general paralysis).** | **Progressive demential** ***states.*** | — | — | Frequently albuminuria. | Wassermann generally positive. | — | — | Lymphocytosis. |

A practical outline of the commoner causes of coma will be found on the following page.

| | History. | Hemiplegia. | Fever | Urine | Blood. | Blood-pressure. | Kernig's Sign. | Cerebrospinal Fluid |
|---|---|---|---|---|---|---|---|---|
| **Uremic.** | That of nephritis and of *arteriosclerosis.* | Occasional, perhaps *transitory.* | Exceptional. | Albumin Casts Reduction of urea. | **Blood urea exceeding 1 gram.** | *High* blood-pressure. | *Exceptional* | Nothing characteristic, but pressure high Low urea content. Sometimes hemorrhagic. |
| **Apoplectic.** | Syphilis Arteriosclerosis Interstitial nephritis | **Constant.** Pupils unequal or dilated Contracted in presence of ventricular hemorrhage | **Frequent.** | Albuminuria frequent. | *Urea content sometimes raised* Wasser-*mann positive in* syphilitics | **High blood-pressure.** | — | |
| **Alcoholic.** | Intemperate habits | — | — | Albuminuria exceptional. | — | — | — | — |
| **Epileptic.** | Previous seizures. | — | | — | — | — | — | — |
| **Diabetic.** | Long standing diabetes (polyuria, polydipsia, and polyphagia) Recent digestive disorders, epigastric pain, emaciation. | — | — | Sugar Acetone Diacetic acid Hyperacidity Special well-*known odor* of breath (chloroform, apple, or acetone). | — | Occasionally high blood-pressure | — | — |
| **Infectious.** | **Causal infection.** (Typhoid, malaria, *jaundice,* etc.) | — | **Constant,** more or less characteristic. | Frequently albuminuria Urobilin. | Leucocytosis. Serum-*diagnosis,* etc | Habitual low blood-pressure | Exceptional. | |

Combinations of these forms may, of course, occur, the combinations, alcoholism with uremia, alcoholism with apoplexy, alcoholism with acetonemia (diabetes), and uremia with apoplexy being the most frequent.

These forms undoubtedly make up over 95 per cent of the cases of coma seen in general practice

The remaining 5 per cent. consist chiefly of:

Infectious and post-infectious coma (lobar pneumonia, typhoid fever, infectious jaundice, malaria, and puerperal infection).

Post-traumatic coma (fracture of skull).

Inflammatory, meningo-encephalitic coma.

Toxic coma (opium, morphine).

Neoplastic coma (brain tumors).

Demential coma (paretic dementia).

According to Pierre Marie, brain compression plays an important rôle in the production of post-hemorrhagic coma, and *complete coma is actually dependent upon brain hemorrhage* (*Presse méd.*, June 6, 1914). It may be of service to reproduce here the following practical conclusions, which Marie deems may be formulated in this connection:

In a patient presenting the signs of cerebral hemorrhage who, three hours after the attack, is in profound coma, the question of performing a decompression operation may be considered (an extensive extravasation of blood is in all likelihood present).

In any patient suffering from cerebral hemorrhage who, after having at first presented only a partial coma, falls progressively into a deep coma a few hours or days later, the performance of a decompression operation should be seriously thought of and the decompression carried out as soon as possible (in this instance the condition present is rather a secondary edema).

At this operation a large decompression opening should be made over the *hemisphere on the sound side* and not over that into which the cerebral hemorrhage has occurred.

3. **Is sugar or albumin present in the urine?** The presence of sugar would suggest diabetic coma; in this case the examination should be supplemented by tests for acetone and diacetic acid and by a determination of urinary acidity (see *Technical Procedures*), thus confirming the diagnosis. The presence of albumin would suggest uremic coma, which is confirmed or excluded by determination of the blood urea Where the latter exceeds 1 gram (it exceeded 5 grams in one of the author's cases), *the diagnosis of uremic coma is assured*

4. **Is there high blood-pressure?** A systolic pressure exceeding 220 millimeters (Pachon instrument), especially if associated with manifest cardiac hypertrophy, gallop rhythm, and albuminuria, certainly justifies a diagnosis of uremia with or *without cerebral hemorrhage, arteriosclerosis, interstitial nephritis*, etc The finding of the "blue line" on the gums of a worker in lead will lead, on the whole, to the same inferences.

5 **Is there some manifest evidence of syphilis?** The finding of osteoperiostitis, glandular swellings, a typical eruption, suspicious pigmented scars, etc, may bring to mind the possibility of a specific cerebral arteritis.

6 **A marked reduction in the pulse rate** would suggest Stokes-Adams' *disease, brain tumor, or opium intoxication.*

7. **Examination of the blood** (urea content and Wassermann reaction) *and, in difficult cases, examination of the cerebrospinal fluid* (cytologic study and Wassermann reaction), should, if possible, be systematically carried out. They will often enable the practitioner to decide at once upon a diagnosis of uremia (where the blood urea exceeds 1 gram), of brain syphilis (positive Wassermann), of cerebromeningeal hemorrhage (many red cells *in the cerebrospinal fluid*), *of tuberculous meningitis* (lymphocytosis), of cerebrospinal meningitis, etc.

The more usual causes of coma in private practice are, in the order of their frequency.

*Apoplectic coma* (hemorrhage and softening of the brain).
*Uremic coma.*
*Alcoholic coma.*
*Post-epileptic coma.*
*Diabetic coma.*

**Habitual Constipation.**

1. This may be dependent upon some *local abdominal cause* capable of inducing habitual constipation, either mechanical (pressure or kink) or reflex.

2 It may be due to some more *general* cause.

1. **Local Causes.**—These comprise all abdominal disorders capable of producing pressure on the intestine, kinking, or reflex spasm.

(a) **Pressure**.—Pregnancy, retroversion of the uterus, fibromyoma, or ovarian cyst in women, prostatic hypertrophy in men, and a tumor of the kidney, spleen, or mesentery, or a stricture, cicatricial band, or peritoneal adhesion in either sex, may so obviously be the cause of obstinate constipation as to require no further mention

(b) **Kinks.**—The causal influence of enteroptosis, whether due to loss of fatty support, pregnancy, obesity, perineal impairment, peritoneal bands, or congenital malformations, has always been in some degree realized Systematic fluoroscopy of the abdomen has, however, demonstrated for it a paramount rôle in the production of habitual constipation. Relaxation of tissues, lack of tonicity, and atony of the abdominal wall generally act in conjunction with the actual kink

(c) **Reflex Spasm.**—This is the obvious mode of production of constipation dependent upon painful affections of the bowel or of neighboring organs—anal fissure, hemorrhoids, chronic appendicitis, cystitis, salpingo-oophoritis, pyelonephritis, prostatitis, etc.

2 **General Causes**

(a) The most frequent general causes are, perhaps, simply habit and sedentary life. Education rapidly accustoms the civilized human subject to inhibit on various occasions the impulse to defecate Propriety, occupation, professional necessities, and likewise the low and repulsive conception attaching to the act of defecation, exert an inhibiting influence, consciously or unconsciously, which leads to gradual suppression of the function, constipation being thus established. To these may be added a number of auxiliary factors such as the disgusting foulness of some civil or military privies; the false prudishness—doubtless

# CONSTIPATION.

[Constipatio, *from* constipare, *to squeeze together.*]

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Constipation is characterized by *infrequency* of the intestinal evacuations and an abnormally *hard* consistency of the feces. What are the actual extremes of constipation? No one, to the author's knowledge, has as yet supplied a satisfactory definition in this direction, and none will be rashly attempted here, nor will a complete semeiologic account of constipation be presented. The subject matter will, in fact, be limited to a succinct résumé of the more important clinical forms of the condition.

Constipation appears to depend, in general:

Either upon a decrease of the peristaltic contractions (hypotonia) or, on the other hand, upon spasm.

Or upon a decrease of the intestinal secretions (hypocrinia).

*Or upon an increase of absorption of the intestinal contents.*

Little space will be devoted to **accidental constipation**, the cause of which is, as a rule, easily ascertained

The latter is not true, however, of **habitual constipation**, a "menace to society" much more certainly than is purgation, denounced, not without justification, by Molière and Burlureaux. Much care should be paid to the detection of the cause of this type of constipation, and especially to finding out whether, in the individual case, it is of atonic or spastic origin.

**Accidental Constipation.**

(*a*) Intestinal obstruction: Strangulated hernia; peritoneal bands.

(*b*) Acute abdominal disorders.

1. Appendicitis.
2. Peritonitis.
3. Hepatic or renal colic, etc.

(*c*) Lead poisoning (lead colic).

(*d*) Certain acute disorders of the nervous system: Meningitis, etc.

4. Lastly, various nervous affections, functional (hysteria, neurasthenia, overwork) or organic (tabes, myelitis, etc.), are accompanied by habitual constipation.

The etiologic and pathogenetic diagnosis of constipation is of paramount importance, since by elucidating the causes of the disorder it often supplies direct indications for treatment, as, *e g*, by psychotherapy, massage, mechanotherapy, electric procedures, diet, etc. Whatever be the cause, however, one should always endeavor to distinguish the *atonic from the spastic type of constipation*. This distinction is sometimes self-evident, but in other instances it is difficult or even impossible to make, the two forms coexisting, both as regards time and space, *i.e.*, either following upon one another or being actually present simultaneously at separate points of the digestive tract Fluoroscopy is of the greatest assistance under these circumstances, there is required, not an X-ray plate, but three or four radioscopies carried out at intervals of twelve to twenty-four hours There may thus be detected intestinal kinks, bands, or segmental zones of spasm or atony, inducing chronic intestinal stasis (Arbuthnot Lane's disease)—a condition thoroughly studied in France by Pauchet—capable of giving rise to most varied disturbances (loss of weight, constipation, anorexia, cutaneous dystrophic states, muscular atrophy, impaired nutrition, cardiorenal and respiratory insufficiency, nervous disorders, joint affections, etc ), sometimes curable by medical treatment alone (general hygiene, diet, massage, physical culture, and thyroid, adrenal, ovarian and pituitary organotherapy) ; at others, in a later stage (with established intestinal kink), demanding surgical treatment in the form of intestinal resections of varying extent.

to be deplored, but quite justified—of young girls; the sedentary life imposed by various occupations; the wearing of corsets; lack of physical exercise, gradually leading to more or less marked atony of the muscles of the abdominal wall, and, ultimately, in women, pregnancy and the impairment of the abdominal wall frequently resulting, at least among civilized populations; all these factors account for the fact that constipation, which is exceptional in animals and savages, and but moderately frequent in men, is practically constant in women, particularly those living in cities.

(b) The **diet** of city dwellers, nearly always defective, is another factor, consisting as it does of white bread, meat, fowl, fish, eggs, potatoes, rice, pastes, pastry, confections, cheese, alcoholic beverages, and water—articles which leave too little residue: "Where there is nothing left the bowel, like the king, is bereft of his prerogatives." The same is true, indeed, of a diet insufficient in amount.

(c) **Various metabolic and other chronic disorders** may likewise induce constipation in one way or another

1 Constipation is rather frequent in **neuro-arthritic** cases, comprising those with gout, diabetes, etc.

2. It is regularly present in **diseases leading to asthenia and cachexia**, such as chlorosis, anemia, senility, various cachectic disorders, and infectious diseases. Prolonged recumbency is, furthermore, not a negligible factor in these cases

3. **The atonic, asthenic type of gastrointestinal dyspepsia**, so frequent in women and nearly always associated with ptosis and dilatation of the organs concerned, is another of the commonest causes of constipation, acting through the three combined factors, reduced secretion, lack of tone, and kinks, with the formation of "dead areas" in the digestive tract.

In this connection the term *gastrointestinal dyspepsia* is to be taken *in its broadest sense, comprising motor, secretory,* or secretomotor insufficiency of the digestive tract—stomach, duodenum, and bowel—and of the related glandular organs—the liver and pancreas The marked importance attached, for example, to duodenal dyspepsia as a result of modern investigations is well-known.

in one of 2 groups: (*a*) The convulsions appear in an acute manner (*b*) They are chronic, *i.e*, habitual, occurring repeatedly at intervals of varying length.

### I.—ACUTE CONVULSIONS.

A. **If the patient is a child,** special thought should be given —and, as a rule, the circumstances under which the convulsions appeared will afford a pointer as to the diagnosis—to:

(a) **Convulsions of Reflex Origin.**—Usually without fever:

*Dental*

*Digestive*, particularly due to colic and intestinal parasites.

*Auricular*, due to foreign bodies in the ear or to otitis media.

*Acute otitis media* is one of the most frequent causes of reflex convulsions in children; further, as is generally realized, it is one of the commonest and most often overlooked disorders. *One should never forget to palpate the mastoids and examine the ears in a child with eclamptic manifestations.*

(b) **Convulsions of Febrile Origin.**—Such convulsions often accompany the *eruptive fevers* and are generally not serious

(c) **Convulsions of Meningoencephalic Origin.**—These are associated not only with fever, but also with evidences of meningoencephalitic disorders such as Kernig's sign, headache, vomiting, disturbances of respiration, etc. The marked diagnostic value of lumbar puncture in these cases is familiar to all. There should also be mentioned the convulsions of heat stroke, dependent upon an intense congestion of the meninges and cerebral cortex.

(d) **Convulsions of Neuropathic Origin.**

1. Infantile eclampsia, a very common disorder, is met with in children as a symptom of various diseases, or as a neurosis in which it constitutes in itself the entire disturbance.

2 In the latter form, or idiopathic eclampsia, there is always present an underlying neuropathic condition, or spasmophilia, consisting of abnormal irritability of the nervous system.

3. Spasmophilia is often due to an acid intoxication of the blood following one of a variety of digestive disturbances which gradually induce poisoning of the system.

# CONVULSIONS (CONVULSIVE SEIZURES).

[Convulsio, *from* convellere, *to shake; sudden and involuntary contractions of the muscles.*]

The term **convulsions** is applied to sudden, involuntary contractions of the muscles **Tonic** convulsions consist of contractions of relatively long duration, causing a condition of almost continuous rigidity, combined with shaking movements resulting in only slight displacements of the parts **Clonic** convulsions are made up, on the other hand, of more or less regular, rapidly alternating contractions, resulting in more or less extensive involuntary movements

Some types of convulsions, *e.g*, those of chorea, athetosis, tics. and tremors present such highly characteristic features that their diagnostic recognition is an easy matter. These forms will not be considered in this chapter, nor will reference be made to partial convulsions such as blepharospasm, spasmodic torticollis, writer's cramp and occupational spasms in general *Actual, more or less diffuse convulsions* need here alone be considered.

The only serious mistake in actual observation that can be made is that entailed by **malingering**. Only exceptionally does such an imposition escape detection by an experienced and well-posted observer; the convulsions are always defective in the sense of being pushed to excess—the subject attempting too "dramatic" a representation—or in being unnatural—the subject being a "novice" Yet the author has met with some "accomplished artists" in this connection whose duplicity was exposed only with great difficulty.

The observation of an actual, not an artificial, convulsive state having been made, the second and highly important step is next to be taken, *viz*, that of finding out its cause

In most instances the clinical history obtained from the patient himself or his relatives will enable the physician to place the case

(*d*) They may develop at labor in an albuminuric woman: *Puerperal eclampsia*

(c) **The acute convulsive seizures may occur in the course of some chronic disorder unattended with fever. These are always cases of chronic systemic intoxication.**

(*a*) UREMIA.—This is shown to exist by the albuminuria, high blood-pressure, and enlarged heart, sometimes attended with gallop rhythm; the edema, which, however, is frequently lacking; the practically pathognomonic increase of the blood urea, and the history (headache, vertigo, vomiting, and insomnia)

(*b*) LEAD POISONING.—This is suggested by the subject's occupation (painter, etc.), the characteristic blue line on the gums, sometimes by paralysis of the extensor muscles of the forearm, by the high blood-pressure, and frequently by the history (headache, insomnia, and lead colic)

(*c*) DIABETES.—Here, indeed, the acute manifestations are generally of the comatose variety, in any event, the **urine examination, always indispensable,** though perhaps more particularly so in the comatose and convulsive cases than in others, including tests for sugar, albumin, and acetone, and a determination of the urinary acidity, will point to the proper diagnosis.

(*d*) ALCOHOLISM.—This tends toward many different forms of convulsions, and from various causes Convulsive attacks in the course of delirium tremens, convulsive uremic attacks superimposed on the alcoholism (cirrhosis of the liver or interstitial nephritis), and hystero-epileptiform seizures in neurotic alcoholics.

This is a matter of great practical importance to resident physicians in hospitals, asylums, homes, dispensaries, and to police surgeons.

1. Absence of albumin from the urine and a moderately tense pulse will, in all likelihood, exclude *uremia*, subsequent determination of the blood urea will, if necessary, set the diagnosis straight

2. An alcoholic spree is obviously capable of bringing on an *epileptic attack* in a person predisposed to such seizures. Biting of the tongue during the attack, with resulting drivelling of blood-stained fluid, the incontinence of urine, the violence of the convulsive movements, the initial cry, and the abruptness

4. The acidosis disturbs the metabolism of the lime salts, which may be said to be essential for normal functioning of the nerve cells.

5. In the pathogenesis of eclampsia are also involved changes in certain of the endocrin glands, especially the parathyroids, which lead to insufficiency of these glands.

6. This insufficiency results in eclampsia because the parathyroids are no longer carrying on their antitoxic *rôle* in the system, while there is also disturbed calcium assimilation.

It is certainly a fact that, whatever may be the exciting cause of infantile eclampsia, some individuals are particularly predisposed to it, *i.e.*, that there does occur a *true spasmophilic tendency* based upon nervous disorder with exaggerated irritability of the nervous system and often accompanied by an abnormally acid condition of the body fluids, or acidosis, probably favored by congenital insufficiency of the endocrin glands.

B. **The patient is an adult.**

(a) *The convulsive syndrome results from some* **pre-existing febrile disorder,** such as *typhoid fever* (of the ataxo-adynamic variety), *cholera, malaria* (convulsive pernicious attack), or *epidemic encephalitis.* In the latter instance the convulsive syndrome may be general or localized (hiccup, nystagmus, abnormal movements of the arm or face) and may or may not be combined with manifestations of the oculolethargic category.

(b) **There may or may not be fever,** *but such fever as exists is manifestly accidental and the history supplies the diagnosis:*

(*a*) The convulsions may follow some *injury to the skull* associated with fracture, extravasation, meningeal hematoma, etc.

(*b*) They may follow:

1. A dog bite. *Rabies* ("hydrophobic" seizures, visceral spasms).

2. A contaminated wound. *Tetanus* (trismus; consciousness completely preserved).

(*c*) *They may follow an acute intoxication, premeditated or accidental*

1. *Strychnine,* the type of convulsive poisoning.

2. Opium, cocaine, or theobromine, in which they constitute a much more exceptional event.

## II.—CHRONIC CONVULSIONS.

In this group, similar convulsive attacks have already occurred. A diagnosis has been rendered—correct, wrong, or misleading; in either case, much useful information can be obtained, either from the patient himself or his relatives, concerning his pre-existing condition. In these cases the stumbling-block does not lie in the paucity of information, but very often instead in an excess of contradictory or even incorrect information.

The majority of cases of chronic or recurring convulsions are due to either *uremia, epilepsy, hysteria, diabetes, alcoholism, progressive general paralysis,* or *Stokes-Adams' disease.*

**Uremia** of the convulsive type is a very common disorder, which should always be thought of in the case of a middle-aged or elderly patient or of a syphilitic. Presence of albumin in the urine, repeated finding of a high blood-pressure, and the coexistence of usual signs of chronic uremia (headache, vomiting, pruritus, insomnia or abnormal somnolence, etc.) will serve to orient the diagnosis; blood urea determination will confirm it.

**Epilepsy** should be suspected in the presence of inherited stigmata of the disorder (malformations of the skull, teeth, etc.), unfavorable family antecedents (epilepsy, alcoholism, dementia, or syphilis), and a history of certain disturbances during childhood (arrested development in certain parts, convulsive attacks in childhood, night terrors, or enuresis), and its existence is confirmed by the features of the attack already specified.

**Hysteria.**—A diagnosis of hysterical convulsions is justified only by careful study of the patient's psychoneuropathic reactions, his suggestibility, the observation of an inveterate mythomania, and the special features of the convulsions themselves.

**Diabetes.**—In this disorder the uranalysis, *which should be a routine procedure,* will supply the diagnosis. Yet it should be borne in mind that uncomplicated diabetes never causes convulsive attacks, and that such attacks occurring in the presence of diabetes constitute almost certain evidence of either hyperacidosis or uremia as a complication of the disease. Routine estimation of the urinary acidity and performance of the tests for acetone and diacetic acid (see *Uranalysis*) will settle the question of acidosis and permit of the prevention of coma. Examination for albumin and casts and

of the attack, frequently with bruising due to a sudden fall; observation of inherited dystrophic stigmata, and the profound coma which follows the convulsive attack, are as many presumptive indications of alcoholic convulsions.

3. A *hysterical seizure* may be started by an alcoholic debauch. The absence of profound coma after the attack; the semi-voluntary, semi-conscious character of the movements, during which the patient neither wounds himself nor receives any hard blows; sometimes the incoherence of speech, the resistance offered to passive opening of the lids, the absence of aura, of the initial cry, of tongue-biting, and of relaxation of the sphincters, together with the "artificial," "overdone," "exaggerated," "theatrical" quality of the proceedings which strikes the experienced onlooker—all these favor an hysterical origin of the seizure

But how is it, asks Meige, that ever since most remote antiquity, through the most diverse civilizations, *the clinical picture of the seizure has remained the same?* This is because it is governed by the structure and functions of the nervous system, the reactions of which remain universal and everlasting. Let there be set free, by reason of some momentary inhibition of the upper brain, the subcortical automatisms, and at once the whole chain of the bulbo-spinal centers bursts into action—the *oculomotors* bring about convulsive movements of the eyes, the *masticatory nerves* grind the teeth together, the facial causes the *grimacing facies,* the hypoglossal throws the *tongue* out, and the glossopharyngeal, spinal accessory, and phrenic are responsible for the *outcries, sighs* and *panting respiration* Lower down, the entire gray axis is set in motion, with resulting *hysterical neck, opisthotonos, abdominal distention,* and contortions of the limbs Even the sympathetic is involved, as expressed in *lacrymation, sweating, salivation,* and *frothing at the lips* Thus, nervous anatomy supplies an explanation for the similarity of these manifestations in all countries and throughout the ages.

There still remains to learn why some human beings are more liable than others to these convulsive reactions This, however, is Apollo's secret! (*Presse méd*, April 20, 1921).

4. Uremia, hysteria, and epilepsy having all been excluded, *simple alcoholic toxic convulsive attack* constitutes the remaining diagnostic alternative.

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determinations of the blood-pressure and blood urea will, on the other hand, settle the question of uremia.

**Chronic alcoholism** is revealed by the usual evidences, *viz*, tremor of the extremities, exaggerated reflexes, gastrohepatic and cardiorenal disturbances, and an abnormal "satiny" softness of the skin, particularly over the abdomen. The latter sign, mentioned by Cabot, is actually a very frequent indication of alcoholism; it seems to be especially characteristic in persons engaged in manual labor, whose epidermis is normally thick and rough. Chronic alcoholism gives rise to convulsive attacks only in the event of an acute alcoholic excess or in a hysterical, epileptic, or uremic individual (see above).

**Progressive general paralysis** is detected by a careful inquiry into the changes occurring in the patient's mentality and affective faculties, which the relatives will nearly always describe: *Changed handwriting, which is sometimes illegible; inability to* carry out correctly certain elementary mental tasks such as adding or multiplying figures; peculiar optimism not justified by attendant facts; inability to keep the attention fixed any length of time on one subject, singular absent-mindedness, bizarre and strange actions, and impairment of memory Later, unconsciousness, megalomania, and paralytic phenomena. A specific history is nearly always present.

The period of general torpor or coma nearly always following a severe convulsive attack is usually attended with a more or less pronounced slowing of the pulse rate, which may descend to 60, 56, or 54, without any special morbid significance. Where, however, a convulsive and comatose attack is associated with bradycardia falling below 40, a diagnosis of **Stokes-Adams' disease** may be almost certainly made This appellation should, in the author's estimation, be temporarily retained, as it designates precisely the clinical syndrome, *paroxysmal bradycardia with convulsive and syncopal attacks,* without stating definitely its nature, which appears to differ in the individual cases (see *Arhythmia* and *Slow pulse*).

# COUGH.

Cough is so common a symptom in disorders of the respiratory tract that these two conditions are naturally conceived of as bearing an almost necessary relationship the one to the other and the equation, cough=respiratory disease, is almost inevitably accredited

**Cough of respiratory origin,** *i e*, dependent upon some disturbance in the respiratory tract, is, indeed, the commonest type, representing about nine-tenths of all cases of cough that come under the physician's care Little space need here be devoted to this form, as the practitioner's attention in these cases is always strongly drawn toward the respiratory tract and systematic investigation is thereupon alone required to ascertain the cause.

This does not apply, however, in **cough of extra-respiratory origin.**

Occasionally a patient presents himself complaining of cough, in whom most careful examination reveals nothing wrong in the laryngotracheobronchial tree Usually it is a fatiguing, paroxysmal sort of cough—a cough which is disheartening alike to the patient and the physician, as the ordinary measures of treatment for cough prove a complete failure

For this form the term "reflex cough" has been coined—certainly an improper designation since cough is always reflex, being a defensive reaction of the system to a peripheral stimulus starting nearly always in one of the sensory terminations of the pneumogastric distributed to the mucous membrane from the vocal cords to the terminal bronchial ramifications, but which may originate very differently in different cases.

Such "reflex" coughs, or better, coughs of extra-respiratory origin, can be mastered only by being familiar with their mode of production, by tracing out their cause, and by instituting a strictly causal line of treatment.

**Clinical Features.**—There are a number of special features which impart to cough of extra-respiratory origin a *special clinical aspect.*

It is a *dry* cough, a "useless" cough—meaning by this that it is purposeless, avails nothing, and that it cannot result, unless bronchitis is simultaneously present, in the expulsion of actual sputum; at the most it may sometimes be followed by the ejection of a little mucus or saliva

It is often a *paroxysmal* type of cough, *i.e.*, one ordinarily consisting of a long series of expiratory jerks, brief, occurring at

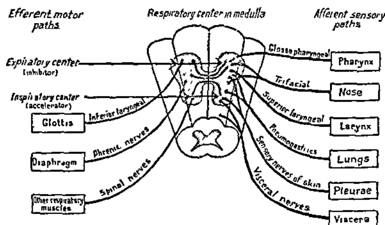



Fig. 601—Diagram showing the afferent and efferent nerve paths concerned in the reflexes of the respiratory tract.

short intervals and separated by relatively few inspiratory movements. It is *particularly distressing* on account of the long duration of the paroxysms and their frequent recurrence.

Lastly, it is a type of cough *which is produced, ordinarily, under certain very special circumstances.* In cases of **pleurisy** (pleuritic cough being, indeed, comprised in this group as the pleura or lung covering is physiologically an extra-respiratory structure), the existing cause is some change in the position of the patient; in some **dyspeptics** it is brought on by food stasis (gastric cough), **worm cough** of intestinal origin is a well-known form in children; laryngologists describe, further, a **nasal cough** and a **pharyngeal cough.** In one of the author's patients, apparently free of all neuropathic taint, endless paroxysms of

cough were started by rather strong odors, such as those of violets and of musk.

**Mode of Production of Cough.**—Physiologic, clinical, and experimental studies of cough lead to a rather definite conception of its *manner of production*. This consists of a peripheral stimulus transmitted to one or more nervous centers and then sent back along centrifugal trunks to the muscles of expiration.

The *nervous center* of coughing is probably closely allied to the respiratory center in the medulla at the nucleus of origin of the pneumogastric, to the floor of the third ventricle and the corpora quadrigemina, and to a few auxiliary centers.

The *centripetal nerve paths* are probably represented by the *pneumogastric;* hence, the almost constant cough noted in affections of the mucous membranes of the respiratory tract supplied by this nerve. The exciting impulse may, however, originate instead in the pleural, pharyngeal, esophageal, gastric, or intestinal branches of this nerve; hence the possibility of having a cough symptomatic of disturbances of these various structures (pleural, pharyngeal, esophageal, gastric, worm cough, etc ).

On the one hand, as with other reflexes, all sensory nerves, including the cranial sensory nerves, may be conceived of as acting like the pneumogastric on the expiratory center, stimulation of these nerves thus being the starting-point of expiratory reflex effects For practical purposes, the *trigeminal* nerve is, after the pneumogastric, in most intimate relationship with the respiratory center, this is the nerve which presides over sensory impressions from the face and the nasal portion of the respiratory passages, in its sphere of action are generally to be sought the exciting causes of cough where examination of the pneumogastric has proven negative An important rôle in the pathogenesis of the cough reflex may also attach to the *glossopharyngeal* Participation of this nerve in respiratory phenomena had been overlooked and unsuspected until it was demonstrated by Laborde in his experiments on rhythmic tractions on the tongue. These experiments showed that the part played by this nerve as an exciting factor of cough, whatever the underlying cause may be, must be duly taken into account, since its sensory fibers

bear an immediate relationship to causes of local irritation that may arise anywhere in the region of the pharyngolaryngeal vestibule.

Sneezing is the most frequently occurring reflex that involves the nasal mucous membrane. Clinically, however, sneezing often precedes cough The physiologic mechanism of the two reflexes is practically the same; most physiologists believe in the existence of a nerve center common to both these acts, and nasal cough is a condition recognized by all clinicians.

The *centrifugal pathways* are many, and need not be enumerated here. Particular reference may, however, be made, as expiratory nerve routes, to the *pneumogastric* (motor nerve to the smooth muscle tissue in the bronchi) and to the *intercostal nerves;* and as inspiratory centrifugal routes, to the *phrenic nerve* (motor nerve to the diaphragm), the spinal accessory supplying the sternocleidomastoid, and the cervical and brachial plexuses supplying the trapezius, scaleni, and intercostals.

Lastly, the manifest inhibitory influence of the will or emotions on cough leads us to the belief that the stimuli emanating from the cerebral centers are conducted, along pathways as yet unknown, to the reflex, automatic centers in the medulla oblongata. This *central action* may, indeed, be *provocative* as in hysterical cough, as well as *inhibitory,* as in the checking of the "useless" cough of consumptives as a result of a peremptory command

**Therapeutic Indications.**—The complexity of the mechanism concerned in the act of coughing and the multiplicity of centripetal routes along which the exciting stimulus may be conducted suggest, *a priori,* the thought that there is not and cannot be any "specific" treatment for the symptom, cough.

Again, cough which leads to expectoration and to evacuation of the bronchi is a *useful cough,* which must be spared or, at most, somewhat reduced This applies to the majority of coughs of respiratory origin. On the other hand, *reflex cough* is generally a *useless* or even harmful manifestation, which must be energetically combatted

Rational treatment should aim: 1. To reduce the sensitiveness of the mucous membrane from which the reflex starts. 2. To

reduce the irritability of the nerve center concerned in the reflex 3. To act if possible on the centrifugal pathways. 4. To inhibit cough through the influence of the higher brain on the bulbar center, *e.g.*, by suggestion.

The first indication, which consists in *influencing the mucous or serous membrane from which the reflex starts*, is generally merged with the causal treatment This is the problem requiring the greatest amount of clinical good sense; it can be solved only after an accurate diagnosis has been attained In a worm-infested individual, a vermifuge remedy will clear up the whole disturbance; in a dyspeptic, proper dieting proves the best remedial measure Where the disordered mucous membrane is accessible to external applications, a local anesthetic will often give excellent results, thus, in two particularly rebellious cases that had proven refractory to most active internal drug treatment, almost immediate results were obtained by placing temporarily in the nasal cavities a wad of absorbent cotton moistened with 1 per cent cocaine hydrochloride solution. In cough of gastric origin, internal use of a preparation containing chloroform and cocaine answers the same indication

The indication which consists in *reducing the reflex irritability of the medullary center* is generally fulfilled by opium and its derivatives Upon opium are based the many official preparations intended for use in controlling cough It is often powerless, if not indeed directly injurious, in the so-called "reflex coughs," except pleural cough The author has had under observation a patient suffering from paroxysmal cough of nasopharyngeal origin in whom a few centigrams of extract of opium brought on paroxysms with such regularity as to exclude the idea of a mere coincidence The value of bromides in these cases has already been amply stressed The so-called "Méglin's pills" have also frequently proven successful in the author's hands

℞ Extract of hyoscyamus seeds ....<br>
Extract of valerian ........ } of each, 0.05 gram<br>
Zinc oxide ........

To make one pill Three pills to be taken daily, morning, noon and evening

The third indication, *viz.*, that of *acting on the centrifugal nerve paths,* is on a more doubtful physiologic plane; yet experience shows that often two fly blisters placed over the course of the phrenic nerve, one in the cervical region, above the clavicle and between the two heads of sternocleidomastoid, and the other at the lower costal margin over the "phrenic point," act most favorably on certain forms of paroxysmal cough, particularly those—and they are frequent—associated with hyperesthesia over the course of the phrenic nerve.

Finally, a *central inhibitory effect* is induced by suggestion during waking hours, the physician asserting before the patient that his cough is purposeless and convincing him of the possibility and necessity of his arresting his cough by a mere effort of the will. The following anecdote, related by Troisier, is quite typical in this connection: "During my visit at Falkenstein, I was seated at the dinner table; I had been given the place of honor, next to the master; a consumptive physician was sitting not far from us He was coughing and coughing without cessation Dettweiler whispered to me: 'You see that physician who is coughing, well, I shall tell him after dinner that he must either cease coughing or take his meals alone in his room, because his cough is unnecessary.' That very evening, at supper, our unfortunate colleague was in his usual place, but he did not cough a single time throughout the entire meal."

On the whole, and by reason of its very commonness, cough is a symptom which, taken alone, is of no great semeiologic significance. It is clinically serviceable, as a rule, only by virtue of the associated symptoms and signs, *viz.*, expectoration, stethoscopic findings, fever, etc.

Yet a few varieties or modalities of cough are deserving of brief mention. From the start, a distinction can and should be made between:

1. The easily executed, expulsive, expectorating cough of respiratory affections in the productive stage, and

2 The distressing, "useless," dry cough of beginning bronchopulmonary infections (before the productive stage) and of extrarespiratory infections (typically pleuritis, pleurisy, or worm cough) The following should also be mentioned:

3. A form of cough which is both muffled (low-pitched) and well-transmitted, "brassy," metallic, usually accompanied by harsh breathing and occurring in the presence of tracheobronchial compression (as in aneurysm, deep-seated goiter, and mediastinal tumors).

4. The nervous cough, habit cough, or "tic cough" of psychopaths.

This occurs mainly under two sets of conditions:

(*a*) As a "defensive" or "disguising" cough, occurring consciously or unconsciously when the patient is embarrassed and desires a momentary interruption in his speech

(*b*) As a "suggested" cough, after the physician, in questioning the patient, has asked him whether he coughs.

In this connection two equally unfortunate suggested interpretations, personal or familial, are to be guarded against:

(*a*) A patient with incipient tuberculosis, desirous of giving the impression that he is not ill, ascribes his cough to habit (a pseudo-psychopath)

(*b*) A psychopathic, obsessed patient, ascribing his cough to tuberculosis, exaggerates the cough (a pseudo-consumptive).

5 The well-known "stridulous," barking, "croupy" cough of croup (diphtheria) or of false croup (laryngismus stridulus).

6 The chronic, habitual, common cough of bronchopulmonary disorders, *viz.*, tuberculosis, emphysema, chronic bronchitis, bronchiectasis, chronic pulmonary congestion, impaired heart-action, etc

7 The so-called "effort cough," generally dependent upon some cardiac disorder and brought on, seemingly, by dilatation of the heart, more particularly of the right auricle. As a matter of fact, in old persons it is often hard to set apart that which, in cough, is due to the heart and that which is due to the lungs, *i e*, to distinguish "heart cough" from "lung cough." Heart weakness, indeed, favors stasis and infection of the lungs; on the other hand, any bronchopulmonary disorder tends to accentuate the cardiac inadequacy and the dilatation of the right heart The cough is then actually and literally of "cardiopulmonary" origin

8. **Postural cough with corresponding expectoration.** The patient coughs especially if he lies on one side, right or left, and the cough is accompanied by copious expectoration. This combination is rather pathognomonic of a bronchial cavity (bronchiectasis or actual cavity), evacuation of which is favored by some definite posture.

9. The **"pharyngeal" cough of smokers and drinkers** is accompanied by hawking and scraping and sometimes by nasal discharge, and is more marked in the morning owing to accumulation of secretions in the nasopharynx during the night.

10. The **cough of pertussis** is easily recognized in the paroxysmal stage with the characteristic crowing inspirations The advantage to be gained if it were possible to diagnosticate whooping-cough early, before the period of the practically pathognomonic coughing spells, is obvious In this connection mention may be made of the work of Modigliani and Villa (*Pediatria*, Apr. 15, 1921) A skin test carried out as in tuberculosis, by intradermal injection of 0.1 cubic centimeter (2 drops) of an autolysate of the Bordet-Gengou bacillus, is stated by them to have given constantly positive results in recognized cases of whooping-cough as well as in incubating cases, and to have been constantly negative in non-pertussis subjects. Their suggestion may well be kept in mind for use during epidemics.

## DELIRIUM AND DELUSIONS. [*from* de, *out of*, lira, *groove*, *out of the groove*, *to wander*.]

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Obviously the aim here sought can merely be to recall the fundamental clinical facts relating to **delirium** and **delusions**, *i e*, those facts with which any practitioner not specializing in psychiatry should be familiar.

To supply a good definition of delirium is not easy. Grasset's definition may here be accepted "Such conditions may be termed delirium as are characterized by disturbances of reasoning power and of judgment, with the reaching of conclusions which the subject believes to be correct and exact" Seglas, quoted by the same author, refers to the fact that delirious concepts may, in different cases, be either vague, indeterminate, or precise and distinct; fixed or changing, polymorphous or uniform; diffuse, monotonous, or systematized (even to the point of "crystallization" or stereotypy), plausible, impossible, silly, fantastic, absurd, incoherent, contradictory, contrasting, antagonistic, etc., and he studies them in succession in such varying forms as deliriums (delusions) of self-accusation, persecution, self-defense, grandeur, hypochondria, negation, enormity, mysticism, eroticism, metabolism, body transformation, etc

For clinical purposes, the large aggregate of different types of delirium may be divided into the three following subgroups, *viz.*, *oniric delirium*, *partial or systematized delirium*, and *delirium of interpretation*.

1 **Oniric or dream-like delirium** is by far the commonest form the non-specialized practitioner has occasion to witness. The patient is then constantly in a condition resembling sleep—he is dreaming The typical delirium of this subgroup is delirium tremens. Lasègue's celebrated definition may be recalled in this connection: "Delirium tremens is a dream which is being lived;" this is the essence of the so-called oniric delirium. The following

description of its essential features is from Régis: "Oniric delirium is an actual somnambulistic state, a second state Like any other second state, it is brought into play through subconscious *or unconscious activity*; it dominates the subject to the point of making him live through and act his subconscious or unconscious life . . . lastly, like a second state, it is always susceptible to hypnotic influence. This form of delirium is, literally, a dream delirium. Indeed, it originates and runs its course in sleep; it is made up of extemporaneous associations of ideas, of *hallucinatory* reproductions of former images and recollections, of scenes of family or occupational life, of visions usually unpleasant, and of strange, impossible combinations which are eminently mobile and changeable or, on the other hand, to some extent fixed, and which *impose themselves more or less completely upon the* patient's belief In its mildest form, such delirium is exclusively nocturnal and evanescent, it ceases upon awakening and reappears only in the evening, either at twilight or not until later when slight somnolence comes on. In a more pronounced form, it again ceases upon awakening, but only incompletely, and recurs during the daytime as soon as the patient shuts his eyes and dozes. *Lastly, in its most pronounced form, delirium fails* to disappear in the morning and continues as such throughout the day, like a prolonged dream."

This is the typical delirium of **intoxications and of acute or subacute infections.** *It is the delirium of the toxic and infectious psychoses in general, of gastrointestinal autointoxications, of alcoholism, of drug intoxications (opium, salicylates, belladonna, etc.), of pneumonia, of typhoid fever, of malaria, of uremia, of eclampsia, etc.*

This, it should be repeated, is the common, ordinary type of delirium which the practitioner sees by far the most frequently.

Its extraordinary frequency in children is particularly well-known; the "dream delirium" is one of the commonest features of children's diseases; it goes hand in hand with fever and accelerated pulse rate in these cases.

2. **Systematized or partial forms of delirium** (paranoia), generally chronic and quite distinct from the preceding types, are made up of *"functional psychopathic states characterized by perma-*

*nent, fixed, and systematically interconnected ideas, developing in a certain direction and following a logical course"* (Arnaud).

Arnaud presents the following classification of these forms:

1. ACUTE SYSTEMATIZED DELIRIUM: Acute paranoia
2. CHRONIC SYSTEMATIZED DELIRIUM: Chronic paranoia

(*a*) *Depressed*.—Delusion of persecution running a systematized course.

Self-accusatory and melancholic delusion of persecution.

Primary systematized self-accusatory delusion

Systematized hypochondriac delusion

(*b*) *Expansive*.—Ambitious (megalomania)

Religious.

Erotic.

The clinical course, now established, of chronic systematized delirium is as follows, according to Grasset:

A. *Period of anxiety or subjective analysis* (hypochondriac insanity), characterized by strange, anesthetic disturbances; the patient is very introspective, and discovers in these disturbances some concealed motive, some allusion to his person or circumstances.

B. *Period of delirious (delusive) explanation* (delusion of persecution, religious delusion, erotic delusion, political delusion, delusion of jealousy, etc); the subject imagines an explanation for his sufferings, for his anxiety, and for the surprising amount of attention which he believes is being paid to him. He discovers the "formula for his delusion"; it is his hallucination which he is interpreting; hence the term "period of delusive explanation"

C. *Period of transformation of personality* (delusion of ambition), from being persecuted, the subject becomes ambitious or megalomaniac, his entire personality is transformed; he becomes a prince, a king, a prophet, or the Deity.

D Magnan recognizes a *fourth period of dementia*, which is a common mode of termination in this kind of psychosis as well as in many others.

*This is the typical delirium of* chronic infections with secondary degenerative changes in the nerve centers.

*Progressive general paralysis*, though much less systematized and coherent than the chronic systematized delirium above de-

scribed, nevertheless presents some of its rather characteristic stages, especially the third and fourth. Two clinical signs point to it particularly, *viz.*, a very special *"euphoric mental puerility"* with amnesia and loss of the autocritical sense (Sicard and Roger), and the characteristic dysarthria with jerky, tremulous, drawling speech, which is unmistakable.

3. **Delirium of interpretation,** a chronic systematized psychosis, founded on *delusional interpretations,* separated in 1902 by Sérieux and Capgras from the group of the systematized delusions, is defined and described by these authors as follows:

"Delirium of interpretation is a chronic psychosis in which the proliferation of manifold interpretations and progressive radiation of a predominant concept result in the organization of a complex delusive romance which may lead to variable reactions. Delirium of interpretation is a constitutional psychosis, the origin of which is to be sought, not in the action of some toxic agent, but in a *psychopathic predisposition, in developmental anomalies of the cerebral centers which hold in dependency perversions of judgment, gaps in the critical sense, and disturbances of affectivity; they are essentially the result of a congenital malformation.*

"Whereas some systematized psychoses are based upon predominant and practically permanent sense disturbances, delirium of interpretation consists of a delusional system in which the hallucinations always remain an incidental occurrence and are even, as a rule, entirely wanting. Lucidity and mental activity are retained throughout the disease; weakening of the intellect appears only after a lapse of time, from the effects of senile evolution, some subjects are seen retaining their mental alertness thirty years after the beginning of the mental disturbances The disorder, while incurable, is thus not one attended with progressive dementia.

"When in the presence of a subject suffering from delirium of interpretation, one is first of all struck by his correct deportment; the observer is sometimes deceived by the brilliancy of his conversation and the accurate logic of his reasoning processes, and is rather disposed to consider him at most as a thinker along fallacious lines, with a tendency to look upon all events from a peculiar angle, and to systematize all exter-

nal or internal phenomena upon the basis of a questionable preconcept.

"The imperative need of referring all to his own person and of interpreting everything in a certain direction, and of emitting, on the whole, only affective judgments marred by errors appears as the sole morbid condition in such a subject.

"The mistaken interpretation, the delusion of personal significance is, indeed, the fundamental manifestation of this psychosis. The autonomy of a morbid entity cannot, however, be based merely upon a single sign. Delusional interpretation plays an important rôle in a number of other psychoses and even in simple passional states. To warrant a diagnosis of delirium of interpretation, a whole group of characteristics must be present, *viz.*, (1) multiplicity and organization of delusional interpretations; (2) absence (or paucity) of hallucinations, and their casual occurrence; (3) retention of lucidity and mental activity, (4) progression through gradual expansion of the interpretations; (5) incurability, without terminal dementia"

* * *

The reason that the author has—with Grasset—deemed it proper to recall the main clinical features relating to the various forms of delirium (or delusion) considered very broadly as "disturbances of the reasoning power and of judgment" is that definition and delimitation of the various forms of delirium is a difficult matter; that the types above recalled and described condense into a small compass a number of psychiatric facts indispensable for everyday practice, and that they will bring to mind various fundamental and necessary acts of psychologic discrimination

As a matter of fact, in general practice *the term "delirium" is applied particularly to the common, ordinary form of the disturbance, i.e.,* to confusion of ideas and the presence of mental images associated with mistaken interpretations and often hallucinations—in a word, *to oniric delirium,* for which careful clinical study will always detect some cause, either:

1. Toxic (alcohol, opium, belladonna, salicylates).
2. Autotoxic (uremia)
3. Or infectious (typhoid fever, pneumonia, malaria, etc )

# DIARRHEA.

[*διαρρεῖν, to flow through.*]

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Diarrhea is characterized by the passage of liquid stools.

Only the most practical, fundamental, and essential facts required in the interpretation of this very common symptom can here be presented

In diarrhea, liquid stools are passed with variable frequency. In general, diarrhea seems to depend upon:

Either an exaggeration of the persistaltic contractions (hyperperistalsis, intestinal hypersthenia).

Or an exaggeration of the intestinal secretions (hypercrinia).

Or diminished absorption of the intestinal contents

Clinically, *diarrhea* may be met with under the following circumstances

I. **Lesions of the intestinal walls,** whether there be *irritation or pathologic changes in the mucous membrane,* as is the case in all instances of *toxic, infectious enterocolitis*

A. **Infectious or parasitic enterocolitis.**

(*a*) In this group are included *ordinary acute enterocolitis, typhoid fever,* and *cholera,* of which mere mention is here sufficient. It should be particularly borne in mind, however, that the greater number of instances of common acute enterocolitis are as yet not accounted for, the characteristic clinical complex may be summarized as comprising diarrhea, fever, leucocytosis, and albuminuria.

(*b*) *Chronic parasitic dysenteriform diarrhea with paroxysmal recurrences* requires more thorough consideration As matters now stand, these dysenteriform types of diarrhea may seemingly be classified, for practical purposes, as follows:

*Ordinary colon bacillus dysenteriform diarrhea,* acute or chronic. These cases yield to interruption of feeding, with restriction of the patient to water by the mouth; to castor oil, and to lactose.

*Amebic dysenteriform diarrhea,* acute or chronic Ravaut and Mauté have plainly demonstrated the selective action of emetine and of arsphenamine in these cases.

*Bacillary dysenteriform diarrhea* (acute or chronic dysentery), amenable to irrigations with silver salts and to antidysenteric serum.

*Dysenteriform diarrhea due to trichomonas* (flagellates).

*Tuberculous dysenteriform diarrhea,* with presence of tubercle bacilli.

These causal distinctions, now absolutely necessary as evidenced by the specificity of the methods of curative treatment, can be established only by bacteriologic examination of the stools (see *Examination of the Stools*)

(*c*) By way of a reminder there may also be mentioned here *intestinal cancer* which, however, causes obstruction much more frequently than diarrhea, except where the lesion is situated low down

B. **Toxic enterocolitis.**—In this group are met the *actually toxic and drug forms of enterocolitis* (enterocolitis due to mercury, arsenic, digitalis, colchicum, etc.), and the *alimentary* forms (botulism, etc ).

There is also the *autotoxic* or *diathetic enterocolitis* of uremia, gout, and diabetes.

Some forms of hyperacute gastroenterocolitis result in a clinical picture well described by Lesieur (choleroid state, reduced output of urine, and uremia), which may be accounted for by intense but diffuse and superficial inflammatory lesions of the digestive tract, particularly the small intestine (congestion with hemorrhage, prominence of the follicles).

The bacteriologic basis of enteritis seems to be variable, different combinations of bacteria being found; a constant feature, however, is weakness and insufficiency of the liver and kidneys This constitutional or acquired weakness sometimes transforms the clinical picture of gastrointestinal infection into that of auto-intoxication and azotemia. Hence the appropriate designation "uremigenous gastroenteritis" proposed by Lesieur.

In all such cases the history or the coexisting diathesic manifestations will clear up the diagnosis

II. **Diarrhea of Nervous and Vasomotor Origin.—Enteric** neuroses are very frequent,

Diarrhea upon emotion and the *enterorrhea* of exophthalmic goiter constitute, as it were, an experimental verification of the neuropathic diarrheal flux that may be met with, frequently in alternation with constipation, in the course of the majority of neuroses. Many instances of paroxysmal diarrhea ascribed to dietary indiscretions are really due to this cause.

In most of these cases the cause of the diarrhea remains wholly obscure The physician finds neither infection (no fever nor leucocytosis), nor ulceration (no blood in the stools), nor food poisoning (no dietary indiscretion). The diarrheal flux may supervene even while a most stringent diet is being adhered to, but nearly always appears in conjunction with overwork, insomnia, nervous shocks, or prolonged stress. It is hard for the uninitiated to avoid the conclusion that an intestinal neurosis exists and to suspect as cause a diminution of vasomotor tone due to abnormal excitation of the sympathetic, seemingly evidenced by the low blood-pressure, high pulse rate, excessive emotional susceptibility, general asthenia, tendency to fainting, and vasomotor disturbances (showing a curious similarity to Graves's disease!) There appears to be some physiopathologic relationship between hyperperistalsis and low peripheral blood-pressure, occurring in conjunction with vasodilatation in the splanchnic area.

It seems not unlikely that the so-called "mucous enteritis" is not a true colitis, but rather a spastic enteroneurosis with alternating diarrhea and constipation and excessive mucous secretion

Diarrhea of circulatory origin is equally well-known, *e.g.*, the diarrhea of atrophic cirrhosis, to which Portal referred when he said· "Wind precedes the rain," alluding to the sequence of *tympanites and diarrhea met with in cirrhosis.* The same condition may occur in cardiac, renal, cardiorenal, and cardiohepatic disorders, although constipation is rather frequently observed under these circumstances.

Mention should also be made of the diarrhea, at times profuse, which may follow reabsorption of edemas, hydrothorax, and *ascites, a feature affording* a definite indication for purgation in the presence of these disorders.

**III. Diarrhea of Digestive Origin.**—The causes in this group are complex and varied

Any condition of gastrointestinal dyspepsia, especially if associated with intolerance of fats ("hyposthenic dyspepsia" insufficiency of the liver and pancreas), is almost necessarily accompanied by diarrhea with passage of an excess of fats in the stools (hypersteatorrhea).

Diarrhea of gastrointestinal digestive origin is thus, on the whole, a manifestation of actual indigestion, subdivision of which may be attempted as follows

1. *Botulism, food intoxication, ptomain poisoning*—The many undoubted cases of collective intoxication, as by cream puffs, game, etc, are manifestly produced in this way.

2. *Overeating*—In these cases the digestive limit or capacity is exceeded; this is the well-known indigestion of released schoolboys and soldiers on leave.

3 *Fat intolerance*—An expression of insufficiency of the liver and pancreas

4 *Achylia gastrica*

5. *Pronounced, abrupt discharge of bile* occurring in overactivity of the liver and resulting in sharp diarrhea in the morning after eating

According to Cabot, the **relative frequency of the various causes of** diarrhea is as follows:

1. *Acute enteritis*
   - (*a*) *Cryptogenic,* five-sixths of all cases.
   - (*b*) *Specific* (typhoid, dysentery, cholera, toxic disturbances), one-sixth of the cases.
2. *Chronic enteritis*
   - (*a*) *Cryptogenic,* nine-tenths of all cases.
   - (*b*) *Of known causation* (digestive insufficiency), one-tenth of the cases
3. *Cancer of the intestine.*
4. *Pernicious anemia*
5. *Mucous colitis*
6. *Intestinal neuroses* and *exophthalmic goiter.*
7. *Tuberculosis.*
8. *Fat intolerance.*

Certain signs and symptoms occurring in conjunction with diarrhea sometimes permit of rather accurate localization of the causal disturbance.

The presence of **blood and pus** in the stools (bloody, glairy stools) is characteristic of ulceration in the large bowel, of varying origin (infection or neoplasm).

The presence of **mucus** and false membrane is often characteristic of an intestinal neurosis.

The customary significance of **fat-laden stools** is well-known; steatorrhea points to insufficiency of the liver and pancreas

**Fluoroscopy after a bismuth meal** and *proctoscopy*, moreover, permit of a most valuable direct examination of the bowel (see *Technical procedures*, in *Part II*).

Frequently, indeed, **direct, gross, macroscopic examination of the feces** affords highly serviceable information. *This elementary clinical procedure is just as essential as uranalysis, taking the temperature, or examining the pulse.* The "offending body" must always be sought, or at least the "evidence" or "witness" of it.

The patient, then, should always be ordered to collect and keep the *feces* for examination.

The following features of the stools should be noted:

**Frequency**: Four, 6, up to 100 a day—the latter in the presence of rectal tenesmus, as in dysentery

**Amount**: From a few hundred grams to several liters, as in cholera and choleroid forms of diarrhea, whence the enormous loss of water from the tissues

**Consistency**: Serous, albuminoid (glairy), mushy, pasty.

**Color**: Brown, as in normal stools.

Dark green. Excessive bile content in certain cases of jaundice; in infantile diarrhea, or after administration of calomel

Decolorized, gray, clayey: Obstructive jaundice.

Red or rust-colored: Dysentery

Black, "coffee-ground". Melena, bismuth, or krameria (rhatany)

Colorless, serous, "rice water": Cholera and choleroid diarrhea.

**Odor**: This is always more or less unpleasant.

Exceedingly malodorous: Putrid diarrhea in botulism, putrefactive gastrointestinal indigestion, and in street cleaners, anatomists, and workers in sewage.

Distinctly acid: In gastrointestinal indigestion with fermentation.

No odor in serous stools.

Kind: Ordinary fecaloid type

Bilious.

Serous.

Watery.

"Stony": Intestinal concretions and coproliths; gall-stones

The macroscopic examination referred to will frequently reveal abnormal constituents of the stools:

*Intestinal parasites.* Tenia, ascaris, oxyuris.

*Undigested food* (lienteric diarrhea). Acute indigestion, excessive peristalsis

*Fats (fatty stools)* Fats present in oily droplets, spherules, or larger fatty masses (affections of the liver and pancreas)

*Blood* Red Hemorrhoids

Black Melena

Intestinal shreds Dysentery

Blood-stained, glairy material Neoplasm

*Pus* Infectious or neoplastic enterocolitis.

*Mucus* and *membranous formations*: Mucomembranous enterocolitis, intestinal neuroses

*Intestinal sand* Mucous enterocolitis

*Rice-bodies* Flakes of epithelial cells: Cholera and choleroid diarrhea

The above brief review suffices to illustrate the very great semeiologic value of a mere macroscopic examination of the stools Correlated with the medical history, other clinical manifestations (temperature, general condition, coexisting digestive disturbances, urinary evidences, etc ), and examination of individual organs (liver, stomach, intestinal canal, etc ), it will generally lead promptly to a correct diagnosis.

In puzzling cases it should be supplemented by chemical, microscopic, and bacteriologic examination of the stools (see *Examination of the Feces*), which is frequently indicated.

## DYSPEPSIA. [δυς, *ill*; πέψις, *coction, digestion; disturbance of digestion, particularly gastric.*]

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The term dyspepsia is here employed in its semeiologic sense, and is applied in a general way to any disturbance of gastric digestion complained of by the patient or observed by the physician. The author would not have attempted to deal with this extensive and complex subject—at least under the above comprehensive term—had he not found certain features of it already partly considered in a section of Cabot's "Differential Diagnosis," upon which some of the material which follows is based, and had not his colleague Léon Meunier consented to draw up an authoritative plan of diagnosis for ulcer and cancer of the stomach.

The author's aim will have been fulfilled if, upon reading this section, the practitioner becomes convinced that dyspeptic manifestations, indigestion, and vomiting are in most instances of extragastric origin; that painstaking and complete investigation of all the organs is necessary, particularly in all cases of chronic indigestion, and that in a patient complaining of such disturbances, merely applying the term "dyspepsia" and ordering some indefinite antidyspeptic treatment amount to nothing, or are even worse than nothing.

*The very great majority of the causes of indigestion are unrelated to the stomach,* or at least to any particular disease of the stomach On the other hand, *there is not a single organ in the human body which may not be a source of gastric symptoms.* Nausea, dyspeptic disturbances, the vomiting of pregnancy, uremia, and brain tumor are familiar illustrations of this clinical aphorism. As a matter of fact, the heart and the stomach may with equal frequency and in an equal degree be disturbed by remote and slight organic causes. The stomach is, moreover, as frequently free of any pathologic changes in subjects com-

plaining of dyspepsia as is the heart in subjects complaining of palpitations or in whom tachycardia is observed.

The truly "gastric" causes of indigestion or dyspepsia may practically be reduced to two, *viz.*, *cancer* and *ulcer* Nervous dyspepsia, the gastric neurosis, is of extraordinary frequency; but only exceptionally does it originate in the stomach. The same is true of many other varieties, such as dyspepsia with hyperchlorhydria; dyspepsia dependent upon constipation or symptomatic of appendicitis; gastroptosis, ordinarily the result of general atony with multiple visceroptosis, alcoholic gastritis, etc. In short, most varieties of dyspepsia are not, strictly speaking, of gastric origin, and do not constitute gastric disorders.

What clinical possibilities, then, should come into our minds when a patient complains of gastric symptoms, and of gastric symptoms alone?

1. In the presence of a pregnant woman who has not yet reached the *menopause*, one should always think first of all of a possible pregnancy. As is well known, under these circumstances any symptoms may be observed, from a simple condition of nausea in the morning to the uncontrollable vomiting of pregnancy—as is true, moreover, in many toxic states of the blood, such as alcoholism, uremia, lead poisoning, etc These digestive symptoms occur with such frequency that they may properly be included among the minor evidences of pregnancy. The classic indications of this state should under these conditions be sought, *viz.*, cessation of menstruation, increased size of the uterus, secretion of colostrum, etc

2 Uremia, manifest or latent, is also a very frequent condition, and one present far oftener than it is diagnosticated. Many obstinate dyspeptic disturbances, either mild (nausea, anorexia, aversion to food) or severe (vomiting, hematemesis), originate in this way Uremia should always be thought of in the presence of chronic dyspepsia coexisting with albuminuria, edema, and a definite elevation of blood-pressure, and particularly if blood examination discloses a high content of urea. Especially should it be thought of, *a priori*, in any individual who after passing the fortieth year, and having had a "good digestion" up to that time, loses his appetite, experiences nausea or even vomit-

ing, becomes sallow and loses weight, and in whom examination of the stomach gives practically negative results. The above mentioned evidences—albumin, high blood-pressure, edema, and hyperazotemia—should be carefully searched for, and the appropriate antinephritic treatment will remove all doubt as to the renal origin of the dyspeptic disturbances.

3. **Tuberculosis,** pulmonary or elsewhere situated, may likewise be a cause of many instances of indigestion in the absence of any *internal (cancer or ulcer)* or *external (food or drug poisoning)* gastric cause As is well known, incipient tuberculosis often takes on the appearances of anemia and dyspepsia; anorexia and loss of weight are common in this stage of the disease. In these cases of "cryptogenic" dyspepsia one should proceed, therefore, to a careful investigation in this direction, the temperature being taken morning and evening, careful auscultation carried out in a quiet room, and an x-ray examination practised.

As a matter of fact, the opposite mistake is also made, and an attack of nervous dyspepsia with anemia and loss of weight too often labelled pulmonary tuberculosis without any adequate reason. The fact cannot too often be repeated: One should be a "realist" in clinical work, and like St. Thomas, one must be desirous to come into actual touch, *i e*, to establish by contact with our senses, the sufferings of our patients and the theories evolved in our minds.

Furthermore, tuberculous patients—**apart from the ordinary causes of dyspepsia** to be mentioned later (rapid eating, poor teeth, excessive intake of fluid, etc.), and to which they are subject like other patients—are very often and very particularly the victims of two serious sources of error regarding the stomach, *viz*, **drug intoxication** *(opium, morphine and its substitutes causing* hypopepsia and apepsia; creosote and its derivatives, antipyrin, pyramidon, etc, inducing æ."gastritis medicamentosa"), and **alimentary overwork** the result of ill-considered overfeeding.

4. Numerous cases of indigestion in women are the result of **inanition.** In this connection the author cannot do better than to quote literally from Cabot, having nowhere found a better or more judicious critical exposition of the abuse of dietetic

measures among dyspeptics "This [inanition] comes about as follows: Something, we need not now inquire what, produces an upset of digestion. The patient attributes it to certain food, probably what she took last, just before the attack occurred. Accordingly, in future she omits this article of diet from her bill of fare. The indigestion recurs, an article of diet is again blamed, and something else is cut out of the diet because she thinks it hurts her. So in this way food after food is given up, until the patient gets down to a regimen of slops or their equivalent. We have now a typical vicious circle The patient is ill-nourished because she is dyspeptic, and she is dyspeptic because she is ill-nourished. We can break this circle by forcing her to eat despite grievous suffering. An ill-nourished stomach will complain, yet it must be nourished nevertheless If we can persuade the patient to undergo such suffering, we can honestly hold out the hope that at the end of it she will break her chain, will get back her nutrition, and lose her symptoms. The trouble is that ordinarily the physician does not believe this himself. He has not seen enough cases in which forcing the patient to eat achieves this happy result; but anyone with extensive hospital experience knows that what is called "dieting"—that is, cutting out of one's diet most of the foods that ordinary people live on —is usually a most pernicious process, and leads to a great deal of long and unnecessary suffering Most cases of this type can be cured by nothing in the world but forced feeding.

"The greatest improvement that I have seen in the management of stomach cases in the last twenty years has been the recognition of causes outside the stomach and the successful attack upon these causes. Next to this, the greatest improvement has been through giving up our habits of making strict, narrow diet lists which result in more or less chronic starvation. Whatever we do for a gastric patient, we must not starve him. We must get in food enough to maintain the caloric needs of the body, and the greatest error in the treatment of the past has been the failure to recognize this necessity"

5 Cholelithiasis is a very common cause of paroxysmal pains, very often ascribed to the stomach. After cancer and ulcer have been correctly excluded, one may state that it is almost always

a mistake to attribute really severe pain to the stomach. In other words, the only disorders of the stomach causing severe pain are cancer and ulcer. All the other varieties of dyspepsia run their course with their usual assortment of symptoms and varying combinations of flatulence, heart-burn, discomfort, nausea, sensations of constriction or oppression, and vomiting, but without violent pain.

Gall-stones often induce attacks of pain situated in the epigastrium and not in the gall-bladder region Overlooking of this fundamental fact leads to many mistakes. If the patient has repeated attacks, some will sooner or later become localized in or be referred to the right hypochondrium, but in the earlier stages of the disease such localization is very frequently lacking

Allied to the gastralgia of cholelithiasis are the obstinate dyspeptic phenomena, with delayed pain and eructations, dependent upon *adhesions between the gall-bladder and the neighboring viscera* (stomach, liver, transverse colon, etc.) the result of a *former cholecystitis with pericholecystitis.* These may assume the extremely grave form of pyloroduodenal stenosis, with vomiting and absolute intolerance of food

6 Angina pectoris may be overlooked and treated as a form of dyspepsia when the pain is felt in the epigastrium, is preceded and accompanied by flatulence and eructations, as is frequently the case, and comes on after meals These three observations of a digestive character, especially when made in combination, lead to many mistaken diagnoses of various gastric affections. Determination of the blood-pressure, careful auscultation, and painstaking inquiry relative to the patient's medical history and the factors which brought on the attack will usually reveal in definite fashion the existence of angina pectoris One of the most constant features of angina pectoris is its almost inevitable occurrence under the influence of bodily fatigue and emotion, and its subsidence under rest and quiet Disturbances of gastric origin *do not have this feature* *In the majority of cases* of angina pectoris the pain, even if it starts and culminates in the epigastrium, radiates to the precordial region and sometimes even into the left arm.

Yet, in truth, the diagnosis is frequently a nice matter, at least when based merely on cursory clinical examination. This is due to the facts that: 1. Many discomforts manifestly referable to the stomach, particularly aërophagia and the gastric neuroses, are frequently accompanied by anginose symptoms. 2. The anginose syndrome (see *Precordial pain*) is of very variable origin and gravity, but if it be recognized that true angina pectoris always constitutes an outward manifestation of myocardial weakness or an aortic or periaortic lesion or the—very frequent—combination of these two conditions, the clinical examination should be directed definitely along these lines The objective evidences of myocardial impairment and aortic lesions should be sought, and if such examination proves negative, the angina theory resolutely discarded.

7. Gastralgic crises of tabetic origin.—The possibility of tabes as the cause of violent, paroxysmal, uncontrollable "crises" of sudden onset and disappearance should always be thought of. Indeed, if, in accordance with the precepts presented in the section on systematic organization of clinical examinations, testing of the patellar reflex and for the Argyll-Robertson pupil is never omitted, many unrecognized cases of tabes will be detected and, in consequence, many gastralgias treated as ulcer of the stomach referred to their true, tabetic origin; even more particularly should these procedures be remembered in gastralgia occurring in known cases of tabes Gastric ulcer or cancer may, however, be present in combination with tabes The fact of having detected tabes does not warrant the practitioner's dispensing with an investigation for the signs of ulcer or cancer

As an exceptional condition, brief mention may be made of the possible presence of gastralgia of syphilitic origin resulting either from the so-called "hourglass" shape of the stomach, probably due to a cicatricial band remaining after cure of a specific lesion, and which fluoroscopy after a bismuth meal will reveal *de visu*, or to specific ulcerations, which appear to be uncommon

8. Dyspepsia and gastralgia due to lead occur much oftener than they are diagnosticated. Painters, printers, and workers in rubber are particularly subject to them; on the other hand, they need hardly be thought of in persons who are not manual work-

ers. Any unaccountable dyspepsia or loss of appetite coming on in an individual who handles lead should be presumed to be of saturnine origin. If to the dyspepsia are added colic and marked anemia, and if the gum margins exhibit the characteristic bluish-gray lead line, a mistake in diagnosis is inexcusable In the premonitory stages, however, one cannot do more than presume the existence of lead poisoning; in any case, application of the classical precept "*sublata causa, tollitur effectus*" is in order; abstention from all contact with lead, proper diet, and the exhibition of diuretics and laxatives will bring about rapid improvement if lead poisoning exists. The patient, thus forewarned, should thereafter take whatever precautions or carry out whatever procedures he may deem appropriate

9. **Cancer of the large intestine** *deceives many clinicians*, even when very well posted, when it is manifested, as is often the case, in irregular periods of nausea, pain, and even vomiting, in the absence of any notable or appreciable intestinal symptoms. A bismuth meal or enema followed by systematic fluoroscopic examination will sometimes alone settle the diagnosis

Simple fecal obstruction of atonic and aged patients may, *indeed, give rise to wholly similar manifestations.* The author will always retain a recollection of a patient seen in the late Dr. Landrieux's service, in which he was an intern at the time. This patient, about 60 years of age, cachectic, anemic, of a straw-yellow color, presented upon palpation a tumor of the size of the fist between the umbilicus and the right hypochondrium: digestive disturbances were very marked, consisting of anorexia, *aversion to meats, frequent vomiting, constipation, etc.* Dieulafoy, from whose service the patient had recently issued, had made a diagnosis of cancer of the stomach, which seemed to the author plainly warranted

Months elapsed without any notable change in the situation, when one day, a large dose of castor oil resulted in the evacuation of several chamberfuls of stercoral masses, together with disappearance of the abdominal tumor and rapid betterment in the digestive disturbances

The subject was seen again in subsequent years, and the improvement thus initiated was observed to have been maintained.

10. **Organic affections of the nervous system,** as well as arteriosclerosis, are very often causes of indigestion; the headaches and vertigo which frequently accompany indigestion in these cases should draw the practitioner's attention to the arteries, kidneys, and brain. It should be recollected that sclerous, specific, or neoplastic lesions of the brain may induce for weeks or even months headaches of the so-called "bilious" type ascribed to indigestion or, if unilateral, decorated with the term "migraine." These mistakes are obviated by careful anamnesis, determination of the blood-pressure, uranalysis, inspection of the ocular fundi, and a search for the indications so often overlooked by the patient or masked, *viz.*, paresthesias of the extremities, slight evanescent attacks of paresis, aphasia, mental confusion, convulsive twitches, etc

11. **Alcoholic gastritis,** a common disorder in some classes of society, and not exclusively in the lower strata, is easily diagnosticated if merely kept in mind: (*a*) By observation of the ordinary signs of chronic alcoholism, *viz*, tremor of the extremities, abnormal excitability, various evidences relating to the mucous membranes, etc. (*b*) By eliciting a history of habitual intemperance, which should be carefully inquired into *in any class of society* by minute questioning regarding the use of spirituous beverages (*c*) By the nature of the dyspeptic disturbances, *viz*, anorexia, aversion to food, and especially the vomiting of mucoid material in the morning—a frequent and characteristic finding (*d*) At times by the simultaneous presence of other visceral manifestations directly or indirectly related to alcoholism, *viz*, hepatic cirrhosis, arteriosclerosis, progressive mental deterioration, etc.

12. **A most frequent clinical type,** particularly in women, is that afforded in the following syndrome of gastrointestinal dyspepsia:

Poor or capricious appetite; a sensation of discomfort, *weight, or tension in the stomach during the digestive* period, continuing for a varying period of time; stasis of food in the stomach, manifested clinically, apart from the unpleasant sensations already mentioned, by regurgitation of food several hours after meals and by succussion splash in the empty stomach in the morning; gastrointestinal fermentation, distention after

meals, and eructations. These patients state that they are greatly relieved by belching, and lay much stress on this fact. Generally there is manifest sluggishness of the bowel, with habitual constipation, sometimes interrupted by attacks of diarrhea; mucomembranous enterocolitis is frequently present, as are also disturbances due to reaction on the liver (slight jaundice, painful congestion of the liver, etc ); frequently the patient appeals to the physician on account of disturbed heart action (palpitations, tachycardia, etc ), or for more or less definite nervous disturbances, such as migraine, general malaise, vertigo, headache, general weakness, and psychasthenia

Upon examination there is found almost invariably a relaxation of the abdominal wall, with lessening of the normal tension of the abdomen; palpation gives a particular impression of softness and atony; it excites no defensive reaction or reflex tension Needless to state, ptosis of the viscera is constant, consisting of descensus of the kidney, liver, stomach, or even the uterus

Low blood-pressure is an almost constant finding; the hyposphyxic syndrome is frequently present, and respiratory insufficiency is the rule.

From the purely clinical standpoint, this condition may seemingly be designated as a **hyposthenic gastrointestinal dyspepsia** (hypomotor and hyposecretory), with stasis, fermentation, ptosis of organs, and various reactions elsewhere, *e.g.*, upon the liver, heart, kidneys, and nervous system.

It should be repeated that few clinical types occur as frequently in women as this one This is sufficiently shown by the large number of investigations that have been made on the subjects of visceroptosis, dilatation of the stomach, movable kidney, flatulent dyspepsia, etc, all of which are simply particular modalities of the foregoing major type

In truth, these are cases of inadequate circulation, respiration, or neuromuscular action as plainly as they are cases of digestive insufficiency They are general **hyposthenics**, the functions of all their organs being below normal (see *Hyposphyxia*).

13. Lastly, one should not forget the **reflex dyspepsia of appendiceal origin**, with or without nausea and vomiting.

Having concluded this lengthy, albeit incomplete, enumeration of the possible causes of indigestion and gastralgia, there remain to be mentioned the commonest and most important causes of these conditions, in the author's estimation.

*The first four are functional.*

1. Bad teeth.
2. Rapid eating.
3. Aërophagia.
4. Psychoneuroses and mental depression; overwork.

*The last two are organic*

1. Gastric ulcer
2. Cancer of the stomach.

**Bad teeth**, so exceedingly frequent a condition, particularly among the poorer classes, is a common, manifest, and unfortunately too often overlooked cause of dyspepsia Patients will come to the physician already provided with strict dietetic regulations and most expertly written prescriptions, in whom but one point in the examination has been omitted, *viz*, that of looking at the patient's teeth—an initial and necessary step in any examination of the digestive tract

**Rapid eating** goes hand in hand with bad dentition; the profound and patient studies of Fletcher should be recollected in this connection Re-education of mastication is sufficient treatment for curing a very large number of dyspeptic cases.

The most elementary clinical investigation, indeed, will afford conclusive evidence in support of the two following axioms:

*A person who masticates his food correctly is almost never a dyspeptic* (apart from obvious dietary indiscretions).

*A person who masticates his food insufficiently is always a dyspeptic*

Practical conclusion In all dyspeptics, whatever be the type of dyspepsia, the prescription should begin with the time-honored but often neglected warning *Eat slowly, masticate the food thoroughly, and moisten it well with saliva*

**Aerophagia** is very frequently combined with rapid eating, and gives rise to similar dyspeptic symptoms consisting of a feeling of weight and distention, meteorism, sometimes with cardiac manifestations, dyspnea, palpitation, or even premature

beats, all very easily accounted for by the anatomic relationship between *the fundus of the stomach* and the diaphragm. Observation of the unconscious aerophagic reflex and percussion of Traube's space insure a prompt diagnosis.

**Psychoneuroses, mental depression, and overwork** induce ordinary manifestations of dyspepsia, either chronic or oscillating like patient's "humor" itself. That mental factors powerfully influence gastric digestion and, *conversely, that disturbances of* gastric digestion powerfully influence the patient's "morale" or "humor" is a clinical fact which is well expressed in the old-fashioned word "hypochondria" and the truth of which ordinary observation will easily prove.

The older authors, *e g*, Chomel and Grisolle, considered dyspepsia a neurosis of the stomach. Subsequent organicist, pathologic, and chemical investigations for a long time eliminated this conception which, however, has never been completely abandoned The most recent investigations point back in its direction; unquestionably the nervous system in the widest measure regulates the secretion and motility of the stomach, and the latter is one of the most sensitive organs in the body, upon which react most frequently, through the solar plexus, all causes of nervous disturbance, whether depressive or stimulating.

As a matter of fact the majority of dyspeptics are psychopaths. Bourget estimated that the "dyspeptics through psychic disturbances" made up three-fourths of the practice of physicians specializing in the digestive tract Mathieu and Roux write that nervous dyspepsias are frequent Dubois, of Bern, asserts that "90 per cent. of dyspeptics are cases of psychoneurosis" The latter estimate appears to the author too high, and is probably accounted for by the fact that *Dubois is a neurologist* The *author's experience leads him to conclude, with Bourget,* that psychopathic dyspepsias make up about three-fourths of all dyspepsias.

Disappointments and constant worry exert a marked influence. How many "hypochondriac" employes show restoration of spirits and digestive capacity through mere promotion to a position sought for a long period! How many ladies, terribly dyspeptic, with a daughter to be married off, find their digestive

## Ulcer and Cancer of the Stomach.

### Catheterization of the Fasting Stomach.

| | | |
|---|---|---|
| (a) Food stasis | ........................ | Pyloric stenosis and, if true stasis, cancer of the pylorus. |
| (b) No food stasis. | Wash water contains free HCl | Reichmann (probably pyloric ulcer). |
| | Wash water, with 1 per cent acetic solution, contains chemically demonstrable blood. | Ulcer of the body of the stomach (simple or cancerous). |

### Examination of the Stomach after a Test Meal.

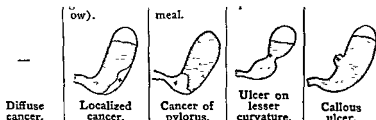
| | | |
|---|---|---|
| (a) Free HCl in excess | Ether capsule dissolved in less than one hour | Probable ulcer. |
| (b) Free HCl reduced toward 0. | Ether capsule not dissolved. | Probable cancer. |

### Examination of Feces after a Milk and Vegetarian Diet.

| | | |
|---|---|---|
| Presence of blood chemically demonstrable | Blood disappears after a few days' rest | Probable ulcer. |
| | Blood still present | Probable cancer. |
| | Blood is present in feces but not in acidulated wash water from stomach | Duodenal ulcer or ulcer on duodenal aspect of pylorus. |

### Fluoroscopic Examination (Principal types)

| | | | | |
|---|---|---|---|---|
| Small, contracted stom- | Filling-defect in stomach | "Amputation" of the py- | Stomach presenting a | Diverticular aspect (ap- |
| | ow). | meal. | | |



| Diffuse cancer. | Localized cancer. | Cancer of pylorus. | Ulcer on lesser curvature. | Callous ulcer. |
|---|---|---|---|---|

### Blood Examination.

Increased antitryptic power of the serum .... .. Cancer.

### Cytologic Examination.

Microscopic examination of the wash water after gastric lavage

Study of centrifugation sediment.

Presence of neoplastic cells . .. .................. ...Cancer.

power suddenly improved at the wedding feast! Unfortunately, in these patients, except in the case of single persons, the physician's rôle is necessarily a rather restricted one.

**Ulcer and Cancer of the Stomach.**—The importance of such diagnoses as those of ulcer or cancer of the stomach is obvious At the author's request, Dr. Léon Meunier consented to formulate a *vade mecum* of the laboratory procedures indispensable in the diagnosis of ulcero-cancerous affections (see the preceding page).

Aside from the classical symptoms of ulcer and cancer of the stomach, *viz.*, late pains, vomiting, hematemesis, cachexia, etc., it is important to be able to make a diagnosis in the earliest stage of these lesions, since often the only effectual treatment, *prompt operation, depends for its practicability upon such early* diagnosis

In the annexed synopsis are presented the various methods of examination which should be availed of when ulcer or cancer of the stomach is clinically suspected, together with the results obtainable therefrom

It should be noted that any one of these procedures is rarely sufficient to afford a positive diagnosis, but that their combined results often lead to a strong probability.

## DYSPNEA. [δυς, *ill;* πνεῖν, *to breathe.*]

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Dyspnea is characterized by a *difficulty in breathing;* it is usually associated with increased frequency of respiration (polypnea) and sometimes, as will be seen later, by changes in the amplitude of the respirations In brief, the essential feature of dyspnea is distress, or even at times pain, attending the respiratory exertion.

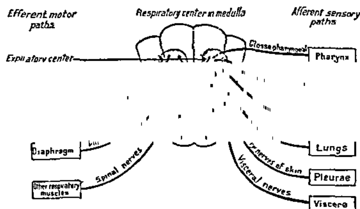



Fig 606—Diagram showing the afferent and efferent nerve paths concerned in the reflexes of the respiratory tract (cough, asthma, etc.). Pathogenesis of dyspneic and asthmatic attacks

All grades of dyspnea are met with, from the *dyspnea on exertion,* appearing only upon more or less marked or prolonged motor activity, to orthopnea, in which extreme dyspnea compels the patient to brace himself against furniture or a window in order to breathe.

Some attacks of paroxysmal dyspnea are known as asthma

It is not within our plan to discuss the physiology of regulation of the respiratory rhythm and the pathologic physiology of dyspnea. It should be noted, however, that the automatic regulation of the respiratory function is effected both by a chemical and a nervous process

1. *Chemical*—The concentration of carbon dioxide in the blood

2. *Nervous.*—Through the pneumogastrics, which embody two kinds of fibers: (*a*) The fibers inhibitory to inspiration and accelerator to expiration which are excited by the expansion or dilatation of the lungs (*b*) The fibers inhibitory to expiration and accelerator to inspiration, which are excited by deep expirations, as in the majority of instances of dyspnea; these are inoperative during ordinary breathing. Any morbid state which leads to an excessive concentration of carbon dioxide in the blood or which by any route directly excites the respiratory center in the medulla, or indirectly through the pneumogastric nerves, is capable of inducing dyspnea in the physiopathologic sense

The **number and variety of the causes of dyspnea** are such that the semeiologic value of this condition is rather limited. In general, however, one may state that there is present:

1. Either a **manifest or latent lesion of the respiratory system.**

Or a **manifest or latent lesion of the circulatory system.**

Or some **grave toxic-infectious state** (**uremia, acetonemia**).

As with most other symptoms, one must likewise reckon with the possibility of **neurotic dyspnea** (due to nervous inhibition).

2 The clinical significance of dyspnea varies in importance and precision according to the degree to which the symptom is independent of other manifestations, *i e*, is observed in the absence of fever, changes in the lungs or heart, acceleration of the pulse rate, and neurotic stigmata One may almost make the assertion that any dyspnea *sine materia,* in the accepted sense of this term, is either a **toxic dyspnea, usually uremic or acetonemic,** or a **neurotic dyspnea.**

3. **Some forms of dyspnea possess** *per se* **a more or less characteristic significance:**

A. Thus, sometimes dyspnea affects more particularly the **act of inspiration,** or, on the contrary, the **act of expiration:**

(*a*) *Inspiratory dyspnea* is characteristic of obstruction of the upper respiratory passages; thus, it is met with in edema of the glottis, croup, diphtheria, laryngeal spasm, tumors of the larynx, foreign bodies of the larynx, trachea, or bronchi, retropharyngeal abscess, Ludwig's angina, or pressure on the trachea (as by intrathoracic goiter, aortic aneurysm, etc.). Particularly difficult

inspiration may be noisy and assume a stridulous quality which is practically pathognomonic of pressure on the larynx or trachea.

(*b*) *Expiratory dyspnea* is frequently accompanied by whistling sounds; as is well known, it is one of the most characteristic features of emphysema and of asthma. It is met with exceptionally in edema of the lungs

(*c*) *Mixed dyspnea* affecting both inspiration and expiration is by far the most frequent form and likewise the least characteristic.

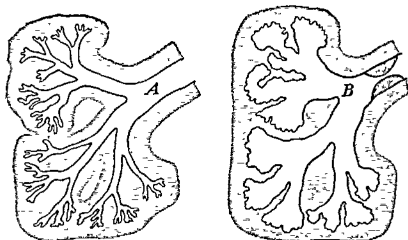



Fig 607—Diagram of a terminal bronchus under normal conditions (*A*) and during a paroxysm of asthma (*B*) (*Abrams*) During the asthmatic attack, the spasm of the circular fibers in the bronchial wall (*B*) causes retention of air in the air-vesicles, difficulty of expiration, and dilatation

B **Kinetic and Static Dyspnea.**—One should also carefully distinguish·

(*a*) *Kinetic dyspnea*, or *dyspnea on exertion, motion, and exercise,* which is merely an exaggeration of a normal event and which appears only upon exertion, such as walking, ascending stairs, exercising, etc The clinical signification of such dyspnea is quite distinct and of great value Any exercise, exertion, or muscular contraction demands increased function on the part of the cardiopulmonary system, which is normally manifested by a temporary acceleration of the heart rate, a rise in blood pres-

sure, an increase in the frequency and amplitude of the respiratory movements, and an increased elimination of carbon dioxide from the lungs. The tachycardia and polypnea induced by exercise are thus absolutely normal phenomena and being such, are not accompanied by cardialgia, palpitation, or dyspnea and subside rapidly after cessation of the exercise. If the exercise taken is too violent or too prolonged, or the reserve cardiopulmonary power of the individual in question is slight, there will occur an *excessive, prolonged dyspnea, palpitation, or tachycardia.*

*Kinetic dyspnea on exertion* is one of the first and most valuable indications of insufficient cardiopulmonary function. The subject notices that he can no longer take a rather prolonged walk or ascend the stairs—as he could previously do without the least difficulty—without experiencing dyspnea The dyspnea on exertion, at first accidental (after a copious meal) or slight (occurring only after relatively violent exercise), gradually becomes habitual and pronounced, requiring for its production only a very moderate amount of exercise

If sought, it will be found present in *all* cases of chronic cardiopulmonary insufficiency, including weakened heart action, anoxemia, inadequately compensated heart affections, and chronic bronchopulmonary disorders such as emphysema, chronic bronchitis, and fibrosis of the lungs.

(*b*) *Static dyspnea continuing while the individual is at rest* either represents the *final stage of the preceding type of dyspnea* or is *the expression of a toxemia.*

Brief mention may here be made of the *influence of posture* and of rest at night.

Dyspnea, of whatever source, is almost invariably increased by the horizontal position, and diminished or relieved by the sitting posture. In extreme cases, indeed, the patients lean forward, with their elbows on their knees, or sit at the edge of the bed or are able to rest only when seated in an armchair Various factors are doubtless operative in this phenomenon, the most important being the pressure on the mass of abdominal viscera and on the diaphragm.

As for the *influence of rest at night,* it may be stated that, with the exception of "dyspnea on exertion" which, as the term implies, passes off after rest, the majority of instances of dyspnea, whether of cardiopulmonary or of toxic-infectious origin, become worse at night. In a certain proportion of cases, this may be ascribed to an unfavorable influence of the horizontal position customarily assumed at night, yet this recrudescence of dyspnea is the rule even in subjects who remain in the sitting position at night. Many plausible explanations have been vouchsafed to account for this fact, *e.g.*, the disturbing influence

**Uremic Coma. Cheyne-Stokes Breathing**

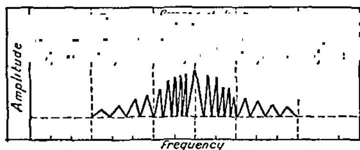



Fig. 608.—Cheyne-Stokes breathing observed during quiet and profound sleep. Systematic observation for eighteen minutes showed ten stereotyped respiratory "cycles" absolutely like that represented above in duration, rhythm, amplitude, and auditory manifestations.

Case 613. Albuminuria, 6 grams to the liter; blood urea, 2.10 grams; blood-pressure, $^{260}/_{160}$; no edema. Other evidences of uremia. Headache, vomiting, flow of viscid saliva, convulsive seizures, etc.

of the night and darkness on the patient's imaginative processes, the period of maximal organic intoxication, a tendency to inhibition of the respiratory center during sleep, etc.

C. **Cheyne-Stokes Breathing.**—Special mention should be made of a particular type of respiration known as the Cheyne-Stokes rhythm, so called after the names of two observers (Cheyne, 1816, and Stokes, 1854) who made a special study of it. It consists of a series of respirations of progressively increasing frequency, amplitude, and noise production (ascending phase), followed by respirations occurring at increasing intervals and becoming smaller and more quiet (descending phase),

after which there appears a stage of complete apnea with cessation of all breathing (period of apnea), the cycle thereafter beginning anew.

This sort of breathing is observed *in its pure form only* if the subject, completely relaxed and unconscious, is in a deep sleep. In the waking state it is always more or less interfered with by mental reactions arising from anxiety, pain, etc.; in the presence of coma, on the other hand, the rhythm is disturbed by *the complications attending this stage, viz., congestion of* the bases of the lungs, partial obstruction of the pharynx and larynx, paralytic phenomena, etc. Yet it is, as a rule, readily detected if the least attention is paid to the respiratory rhythm. The stages of apnea are particularly characteristic.

When the condition is observed in its pure form during *sleep* one is always impressed with the accurate control characterizing the phenomenon, which recurs with mathematical regularity, as in the case herewith illustrated.

Sometimes, though exceptionally, the foregoing rhythm of respiration is present, but without any stage of apnea (Biot's breathing). Such respiration possesses the same clinical significance.

*Cheyne-Stokes breathing* is almost universally looked upon as an indication of serious impairment of the bulbar centers, which have a tendency to "go to sleep" and the activity of which is excited only by such a carbon dioxide stimulation as attends beginning asphyxia.

By far the most frequent cause of it is *uremia,* and the prognosis is usually very unfavorable.

From the standpoint of semeiology alone, the various kinds of dyspnea may be grouped as follows:

| Dyspnea of Respiratory Origin. | |
|---|---|
| Type case Pneumonia. | [illegible] |
| **Dyspnea of Circulatory Origin.** | |
| (a) **Cardiac**<br>Type case **Heart failure.** | Cardiac insufficiency, hyposystoly, hyposphyxia, inadequately compensated heart affections (cardiac pseudo-asthma) |
| (b) **Dyscrasic.**<br>Type case: **Uremia.** | Anemia or anoxemia<br>Uremia or acetonemia<br>Intoxications (certain asphyxiating gases).<br>Febrile disorders |
| **Dyspnea of Nervous Origin.** | |
| Type case **Hysteria.** | Neuroses Neurocardiac erethism<br>Asthma, certain forms of<br>Bulbar affections |

## I.—DYSPNEA OF RESPIRATORY ORIGIN

This is usually obvious, the relationship of cause to effect being in most instances readily demonstrable.

Such is the case in foreign bodies in the respiratory passages; in pressure and obstruction of the pharynx, larynx, or trachea (as in nasopharyngitis, adenoid vegetations, laryngeal diphtheria, tumors of the larynx, and tumors or glandular enlargements in the neck); in bronchitis—especially capillary bronchitis; in bronchopneumonia, congestion of the lungs, lobar pneumonia, pleurisy, pleuropneumonia, etc, further discussion of which would seem superfluous.

These instances of dypsnea of *pleurobronchopulmonary origin* are produced, on the whole, in the same way, *viz, through suppression of a more or less extensive portion of the functionating lung surface,* owing either to compression, as in pleurisy or pneumothorax, to encroachment upon the bronchi, as in bronchitis; to encroachment upon the air vesicles, as in pneumonia and bronchopneumonia, or to inadequate motion of the ribs and diaphragm, as in emphysema, etc

The paroxysmal attacks of dyspnea commonly known as asthmatic seizures, which may, as will be seen, be of very variable origin, hardly exhibit any special differential features ac-

cording to their varying cause; the two essential and characteristic factors are: A *pneumospasmodic disturbance* (the *paroxysmal dyspnea*) and a *secretory disturbance* (the *catarrhal condition*). No lengthy description of these features need here be given. Their essential characteristic is paroxysmal dyspnea, together with bradypnea or slow breathing. It is in tracing the underlying cause of the asthmatic state that the therapeutist is called upon to display the highest degree of clinical common sense and carry out the most painstaking inquiry, for upon this investigation curative treatment mainly depends. No form of clinical investigation is more difficult, and while at times the physician may succeed in detecting the exciting factor at once, more often a prolonged and painstaking search is required, since "anything may happen in asthma, and even, in the presence of certain bizarre manifestations, to be skeptical would be a mistake" (Brissaud).

In practice, the following 6 groups of causes, which are by far the commonest (Moncorgé), should be examined for: 1. Neuroarthritism 2. Causes relating to the lungs. 3. Cardio-arterio-renal causes. 4. Gastro-hepato-intestinal causes. 5. Toxic-infectious causes. 6. Nasal hyperexcitability. As may be noted, according to this almost the entire field of internal medicine will have to be gone over

Mention should be made again of the fact that chronic bronchopulmonary affections—particularly emphysema, asthma, and chronic bronchitis—necessarily react upon the right heart, and that at a more or less advanced stage of the disorder dyspnea is as much of cardiac as it is of pulmonary origin

It should be recalled, furthermore, that many lung manifestations are merely a symptomatic expression of some general morbid condition, such as uremia or cardiac inadequacy; this is true, *e.g.*, of passive congestion of the bases of the lungs, of acute and subacute edema of the lungs, of many instances of chronic bronchitis, and of many asthmatoid conditions *Cardiorenal insufficiency is at the bottom of a very large number of acute and chronic respiratory manifestations.*

For these various reasons, the semeiologic study of dyspnea associated with some definite localized disorder in the respiratory

tract necessarily indicates a careful examination of the heart and kidneys, both for diagnostic and for prognostic purposes.

## II.—CARDIAC DYSPNEA

As for so-called "cardiac dyspnea" one cannot do better than quote the succinct, profound, and clinically excellent presentation of the subject by Ribierre:

"From the start, *dyspnea on exertion* (appearing upon climbing stairs, steeply inclined streets, etc ) is accompanied by *painful sensations* behind the sternum and in the epigastrium, and to these painful sensations is attached from the outset an element of angor, although they are evanescent and quickly disappear at rest.

"Next there is *decubital dyspnea,* coming on suddenly just before or during sleep and likewise accompanied by precordial *anginose pains,* frequently radiating to the back, shoulders, and arms Sometimes the dyspneic element clearly predominates over the painful element; there exists then an asthmatoid dyspnea or, in accordance with the rather questionable term sanctioned by usage, a cardiac pseudo-asthma

"When one takes into consideration the special features attending these painful manifestations, *angina pectoris* at once comes to mind. Is it wise, on account of slight symptomatic differences relating to the duration and severity of a symptom, to perpetuate former misconceptions and establish a definite distinction between these anginose pains, constituting a supposed *angina minor* (which ought not to be fatal!), from the true angina, which ends fatally? Here again, the subsequent course of the case will bring out the true state of affairs Not infrequently, indeed, there are seen to appear, in subjects who had previously exhibited only this relatively mild syndrome, the major phenomena of insufficiency of the left ventricle, *viz.,* a most typical *angina pectoris,* and also *edema of the lungs,* of which no detailed description is here required Since the investigations of Merklen, it is no longer possible to ignore the close relationship existing between the painful dyspnea of high pressure cases and angina pectoris and edema of the lungs, nor the relationship of these syndromes with left ventricular insufficiency."

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the classical picture of complete cardiac insufficiency, involving both the right and left auricles and ventricles.

Lastly, it should be mentioned that in simple fatigue of the myocardium there may be observed in a very mild form a kind of dyspnea suggesting Cheyne-Stokes breathing (Fig. 611).

Cardiac asthma.—Cardiac asthma consists, according to Merklen's definition, of a *paroxysmal dyspnea complicating disturbances of pulmonary circulation and of the cardiac function.*

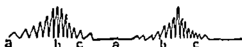


Fig. 610.—Cheyne-Stokes breathing *a*, apnea, *b*, ascending phase, *c*, descending phase

Cardiac asthma generally occurs in subjects presenting unmistakable evidences of insufficiency of the heart, such as difficulty in walking, dyspnea on exertion, dyspnea in recumbency, habitual breathlessness, and attacks of threatened pulmonary edema manifested by slightly reddish albuminous expectoration, gallop rhythm on auscultation, reduced blood-pressure, etc,

At times there are mild, incipient seizures occurring occasionally, either at the moment of retiring, or during sleep, or even

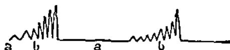


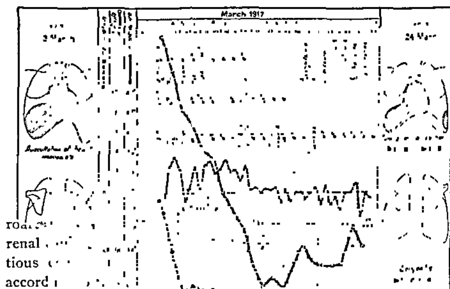
Fig. 611—Dyspnea of myocardial fatigue and general exhaustion *a*, apnea, *b*, ascending phase, descending phase absent.

several times in a single night. These are the result of a weakness of the myocardium which is favored by the reduction of circulation that naturally occurs during sleep and which passes off on awakening. Sometimes these attacks recur as soon as the patient attempts to fall asleep again Occasionally, after taking cold, or a heavy meal, or some emotional impression, there is a severe, dramatic attack with anginose manifestations

Oppression is greater in cardiac asthma than in nervous asthma; this is because of an interference with pulmonary circu-

The above word picture is particularly applicable to the *cardiac dyspnea of high pressure, aortic, cardiorenal, and nephritic cases.*

In *insufficiency of the right ventricle, dyspnea on exertion* sets in gradually, without concomitant precordial pains. Then *the dyspnea becomes continuous,* making it impossible for the patient to remain in dorsal decubitus and being progressively supplemented



The attacks of asthma are, as Merklen puts it, prepared for by pathologic changes or functional disturbances of the myocardium, the chief causes of which are tobacco abuse, alcoholism, overwork, and impaired coronary circulation They are directly brought on by any factors tending to produce or increase the dilatation of the heart. The most important of these causes are those which induce peripheral vaso-constriction—exposure to cold, emotional impressions, indiscretions of diet, excessively long tramps or prolonged work, sexual excesses, intercurrent diseases, influenza, and pneumonia.

Thus, the subject of *cardiac dyspnea* would be a fairly lucid one were it not rendered obscure by the cardiac neuroses, *i.e.*, those individuals who, apart from any acute or chronic organic lesion of the heart or its separate layers (endocarditis, pericarditis, myocarditis), and even in the absence of any true—*e.g.*, congenital or constitutional—myocardial weakness, or of any known disease of the nervous system, suffer from some symptom-complex preeminently involving the heart As a matter of fact, it is these cardiac neuroses which are accompanied by the most numerous and distressing cardiac or pseudo-cardiac symptoms, the most important of which are dyspnea, choking sensations, or angor with radiation of pain to the arm and neck (nervous angina, etc ).

Differentiation of cardiac neurosis and organic disease is not always an easy matter Auscultation may be difficult and misleading; various types of arhythmia—extrasystoles, nodal rhythm, etc —may be observed in either condition; the same is true of the customary hypertrophy of the left ventricle and even in greater degree of the subjective manifestations, such as dyspnea on exertion, sensations of constriction, or even an anginose syndrome, palpitations, phrenocardia, etc Nevertheless there are some differential points

(*a*) The first and most important is, perhaps, the *neuropathic substrate* over which cardiac neurosis always runs its course. The cardiac symptom-complex above referred to is but a portion of the neuropathic picture, always more or less distinct and generally supported by heredity and the extracardiac neuropathic manifestations, particularly digestive and mental.

lation, which, however, is not always readily detected. Frequently, indeed, there is elicited on auscultation merely an exaggeration of resonance, due to a species of acute emphysema of the lung brought on by spasm of the muscles of respiration. In other instances, there develops a more or less extensive pulmonary edema, generally confined to the bases of the lungs. Serous transudation may be sufficiently free to lead to the expectoration of albumin-laden sputum stained with blood—a process which brings some relief to the patient. In such instances, cardiac asthma and acute edema of the lungs coexist. Finally, the tracheal râle may be noted in the absence of râles in the lungs.

Cardiac asthma may be *complicated with angina pectoris*, and this complication is met with particularly in subjects poisoned by tobacco, suffering from sclerosis and atheroma of the coronaries, or subjected to a severe degree of overwork. The left *heart reacts to the distention in common with all reservoir-like* organs with muscle tissue in their walls, *e g*, the bladder; hence the pains felt by the patient. These pains cease when the heart is dilated.

During an attack, auscultation of the heart is frequently impracticable Sometimes one may note gallop rhythm, which is an expression either of cardiac insufficiency in general or a mitral insufficiency from dilatation, which disappears as soon as the heart has become restored to its normal dimensions.

Cardiac asthma may eventually terminate in fatal syncope. In such instances, the extremities become cold, there is incontinence of urine and feces, the patient is covered with sweat, his sight becomes dim, and death supervenes very quickly. In many instances the fatal termination occurs only after the lapse of one or two hours. Some patients pass into a comatose state. As a rule, however, the case does not die if the necessary measures are taken in due time, and the physician always has reason to hope for termination of the attack, even under what appear to be most unfavorable circumstances

Cardiac asthma is the result of sudden insufficiency of the left ventricle; it is due to an abrupt turn for the worse in a latent heart disorder.

The attacks of asthma are, as Merklen puts it, prepared for by pathologic changes or functional disturbances of the myocardium, the chief causes of which are tobacco abuse, alcoholism, overwork, and impaired coronary circulation. They are directly brought on by any factors tending to produce or increase the dilatation of the heart. The most important of these causes are those which induce peripheral vaso-constriction—exposure to cold, emotional impressions, indiscretions of diet, excessively long tramps or prolonged work, sexual excesses, intercurrent diseases, influenza, and pneumonia.

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(*b*) The second is the *frequency* and *severity of the symptoms occurring at night, viz.*, insomnia, anxiety, dyspnea, and even angor and cardiac pseudo-asthma, incomparably more frequent, impressive and seemingly more "dramatic," as a rule, than in cases of organic heart disease These nocturnal psychosomatic disturbances are very definite in neuro-cardiac cases.

(*c*) *Neuro-cardiovascular instability and mobility* constitute the external evidences of excessive nervousness, the pulse frequency and blood-pressure being affected to a surprising extent by the slightest disturbing causes. The same is often true of the auscultatory signs, which are far from being as relatively fixed and constant as those present in organic disease.

(*d*) *Absence of the customary etiologic factors of organic heart disease* is, as a rule, noted, *viz.*, rheumatic, typhoid, syphilitic, diphtheritic, or other infections, plethora and auto-intoxication, gout, uricemia, etc.

(*e*) Lastly, the *circulatory functional test* (see the *Circulatory system*) clearly demonstrates the heightened vasomotor reactions and the usually considerable margin of safety in the reserve power of the myocardium.

### III.—DYSCRASIC DYSPNEA.

One of the most salient features of this type of dyspnea is that of being for a long period *sine materia*, and the fact that neither the time-honored and already somewhat obsolescent routine examination of the circulation, nor an examination of the respiratory system, reveal any disorder or organic disease which might plausibly account for it.

Whereas, however, the objective cardiopulmonary evidences and the classic symptoms already mentioned are ordinarily lacking, and while such dyspnea, considered from this already antiquated standpoint alone, are *sine materia*, such is far from being the case if they are studied and listed with the assistance of the modern methods of examination.

Actually, most cases of this type of dyspnea may, clinically, be placed in one of the three following groups:

1. **Anoxemia.**—At bottom, in these cases, there is a functional cardiopulmonary insufficiency unattended by any recog-

nized disease, but in which modern means of investigation generally elicit: (1) Insufficiency of respiration (the spirometer showing a breathing capacity below 2 litres in an average subject). (2) Low blood-pressure, with the systolic at 120 millimeters or less and the pulse pressure at 15 to 35 millimeters. (3) Increased blood viscosity, 4.5 and upwards, or at least a relatively high viscosity, 4.2 and upwards. Fluoroscopy and orthoradiography demonstrate this lowered functional and nutritive state of the heart and lungs even more rapidly, showing microcardia (the small "drop" heart), insufficient breathing particularly manifested in a relative reduction of the excursions of the diaphragm, reduced clearing of the lung margins during inspiration, and the presence in these parts, especially at the bases of the lungs, of zones which hardly exhibit any clearing at all during inspiration. These are cases of *hyposphyxia* (see *Low blood-pressure*).

2. **Uremia.**—Whether manifest or latent, uremia is perhaps, after hyposphyxia, the commonest cause of dyscrasic dyspnea. Apart from the customary but often misleading evidences of uremia, *viz.*, headache, nausea, itching, cramps, numb fingers, sleeplessness, albuminuria, etc., there are now available certain practically pathognomonic signs, one of which in particular, *viz.*, hyperazotemia, or an excessive amount of urea in the blood—0.60 or more—should be examined for by determination of the blood urea in all suspected cases. High blood-pressure, exceeding 200 millimeters, the presence of traces of albumin, and the passage of an excess of urine of low specific gravity at night, in a dyspneic subject, are in themselves almost pathognomonic of azotemia, especially if the dyspnea assumes at times the so-called Cheyne-Stokes character. In uremic poisoning the azotemia is much more active in causing dyspnea than the chloridemia, which appears to act rather in a mechanical manner (through hydremic plethora and edema of the lungs).

3. **Acetonemia.**—As is well known, some cases of dyspnea, in which indeed the prognosis is highly unfavorable, are associated with the presence of acetone in the urine and with urinary hyperacidity (see *Uranalysis*). This is the case in the dyspnea, sometimes fatal, witnessed in the final stages of some cases of diabetes. The precise mode of production of this kind of dyspnea is

still being widely discussed. One need here merely recall that such dyspnea is always accompanied by *acidosis*, which is expressed, among other manifestations, in *urinary hyperacidity*, and by *acetonemia* (test for acetone), and that it is met with chiefly in the terminal stage of diabetes (*diabetic coma*).

From the above the reader will have gained an idea of the importance of chemical studies of the urine and blood in the presence of dyspnea unattended with definite cardiopulmonary pathology.

As possible causes of dyscrasic dyspnea may be mentioned:

1. Some forms of *intoxication* Asphyxiating gases.
2. Certain *febrile conditions*

### IV.—DYSPNEA OF NEUROPATHIC ORIGIN.

This type of dyspnea is mainly represented by **hysterical polypnea**, which can, as a rule, be readily differentiated by means of the following features:

1. There is very marked *polypnea*, the rate of respiration reaching or exceeding that noted in the final stages of cardiopulmonary diseases.

2 It occurs in the absence of any marked or even appreciable lesion of the heart or lungs, cough is practically absent and expectoration nil

3. Such *polypnea may be greatly reduced or even completely* removed by distraction of the patient's attention, as by an interesting conversation, it may cease while the patient is answering questions, in any event, it is never continuous, but is paroxysmal, coming on in distinct attacks.

4. This syndrome is present in conjunction with the psychopathic state characteristic in these subjects, evidenced by suggestibility, distortion of the truth, mythomania, etc.

5. There is absence of the other signs of true dyspnea: No cyanosis, little or no acceleration of the heart rate, no reduction of urinary output, no edema, no azotemia, etc.

# EDEMA.

[οἴδημα, *from* οἰδεῖν, *to swell.*]

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Edema consists of a serous infiltration of the subcutaneous cellular tissues or of visceral tissues, as in edema of the meninges —involving the pia mater,—of the lungs, etc The ordinary edema of the subcutaneous cellular tissues will here alone be considered. Its characteristic feature is the lasting depression in the edematous region made by any form of pressure (depression from pressure with the finger, grooves from folds of the clothing or pillow, depression with central groove upon pinching, depression *en masse* by lying on an edematous region, etc.).

While sometimes obvious where the swelling has caused a marked change in the shape of an affected part, careful examination is frequently necessary for the detection of edema: In the lower extremity it appears more particularly in anterior pretibial tissues, in the region of the malleoli, and on the posterointernal aspect of the thigh In some subjects the lower lids constitute a seat of election, exhibiting a characteristic prominence In patients confined to bed one should not forget to examine the sacral region and the posterointernal aspect of the thighs In persons who still leave their beds and walk about, it often appears only after they have been standing for a more or less prolonged period, so that, while absent in the morning, it reaches its maximal degree in the evening before retiring. At the very beginning it may sometimes be elicited only by noting an unusual sensitiveness of the cellular tissue to palpation, constituting a species of pre-edematous hyperesthesia

In established edema, the skin is, as a rule, tense, shining, and pale (*white edema of kidney cases*); in heart cases, with impaired heart action and hyposphyxia, the skin may assume a livid, purplish tint (*blue edema of heart cases*); in inflammatory edema, as in suppurative lesions and lymphangitis, the color ranges from pink to red (*red edema of infection*); in hard, chronic edema the color

may become darker, a bronze shade appear, and pigmentation of the skin occur (*bronzed edema of chronic cases*).

The usual consistency of edematous tissues is soft; the finger sinks into them as though into butter; if the illness is prolonged, *and the edema becomes chronic, the cellular* tissue undergoes sclerosis and the condition becomes a hard edema This is the case, in particular, in the chronic edema of cases of varicose veins, of lymphangitis, of myxedema, and of elephantiasis.

* * *

The cause of edema is often plainly apparent, the case being either one of **heart disease**, with cyanosis, dyspnea, oliguria, an overburdened disordered heart, and arhythmia; *or one of* **chronic nephritis**, with pallid skin, gallop rhythm, and more or less pronounced albuminuria; or one of **malignant disease of the stomach**, greatly emaciated, cachectic, and with the lower extremities enormously swollen; or one of **puerperal phlebitis**, with fever, leucorrhea, etc.

Sometimes, and even frequently, more than a single cursory examination is required. While an experienced clinician will almost always be able to get right to the point by virtue of practical knowledge previously acquired, a less experienced practitioner would do well to carry out the following funda*mental steps in the process of diagnostic analysis:*

Edema may be due to either a local or a general cause.

**The local causes** *of edema should be examined for:*

(*a*) **Skin and cellular tissues.**—*Infections* and *toxic infections of these tissues.*—Furuncle, carbuncle, *lymphangitis*, abscesses, and erysipelas are always accompanied by local edema. Are not the three cardinal signs of infection, known since the early historical ages, *swelling, redness, and pain? Mere recollection of* the fact is sufficient. Allied to these conditions are certain disorders, rare in the Western countries, involving the lymphatic tissues (pressure or obliteration, of bacterial or parasitic, *e.g.*, filarial, origin) and causing edema of the type of elephantiasis

*Certain poisonous* bites, mosquito or snake bites, etc.—produce a similar effect.

Acute eczema may be attended with edema.

For the sake of completeness the post-traumatic form of edema, particularly following fractures, may be referred to.

(*b*) **Veins.**—*Phlebitis,* whether primary or secondary, and whether infectious (as is the rule) or dyscrasic (as is much less common) or neoplastic (as is much rarer still), is nearly always attended with edema confined to the affected limb. It is seldom wanting, even during the pre-obliterative stage.

Pain more or less limited to the course of a vein, sometimes the feeling of the vein as a characteristic cord, the observation of more or less extensive edema, the knowledge of some previous infectious disorder (puerperal infection, typhoid fever, rheumatic fever, etc.) or of a gouty tendency or a tuberculous or neoplastic affection causing cachexia (*phlegmasia alba dolens*) are the very features required for a diagnosis of phlebitis.

**General causes.**—The above causes being, as will have been noticed, easy to exclude, the various possible general causes remain to be considered.

In the presence of a definite, manifest edema, one should think *mainly* of the three commonest causes, *viz.*, *cardiac, renal,* and *hemic* (dyscrasic), and *secondarily,* the former having been excluded, of the three exceptional causes, *viz.*, *hepatic, nervous,* and *dystrophic.*

I. **Cardiac edema.**—This is a characteristic feature of *cardiac insufficiency* or *hyposystoly.* The diagnosis is obvious in the stage of *asystoly* or complete decompensation: Marked edema, constant dyspnea, tachy-arhythmia, oliguria, edema of the bases of the lungs, passive congestion of the liver, cardiac dilatation, and frequently, the observation of a definite heart lesion

*At the outset* of heart disease one should look carefully for the *minor evidences of impaired heart action, viz.*, dyspnea on exertion, nycturia, orthostatic oliguria, vesperal edema of the lower extremities, and persistent pulse acceleration after exertion (see *Functional tests of the circulation*)

In these cases water retention and chloride retention are often closely parallel

II **Renal edema.**—This is perhaps the most frequently encountered of all the varieties of edema; often it is present in association with the preceding variety.

Here again, the diagnosis is sometimes obvious, as in *acute nephritis* where it develops suddenly, accompanied by fever, pronounced albuminuria, and even hematuria, and in *long-standing, established chronic nephritis.*

In early cases a special examination for it is frequently necessary, edema being looked for in the lids, the cheeks (as shown by map-like formations on the face, found in the morning and due to pressure by folds of the pillow), and the malleoli. Its appearance is generally preceded by a *stage of pre-edema, or better, of internal, invisible edema,* demonstrated by periodic weighing of the patient and by determination of the chlorides, which will indicate in a parallel and synchronous manner any increase of weight and retention of chlorides.

In all instances the physician should carry out a systematic, complete investigation of the renal functions (see *Examination of the urinary system*), including examination for albumin and casts, and determination of the amount and rhythm of urinary excretion, the systolic and diastolic blood-pressure, elimination through the kidneys, chlorides in the urine, and blood urea. Such an investigation is particularly necessary in the course and during the aftermath of infectious diseases, especially scarlet fever.

Renal edema is, as is well-known, related mainly to retention of the chlorides

III. **Hemic or dyscrasic edema.**—This form, the mode of production of which is as yet quite uncertain, is the one met with in *anemias* of the pernicious type and particularly in the final stage of *cachectic disorders* (tuberculosis and malignant growths). Whether it be true or fiction, it is related that Trousseau, who had been suffering for a long time from digestive disturbances resulting in loss of strength and pain, declared to Dieulafoy, his pupil, that he was afflicted with a growth of the stomach and forecast an early, unfavorable termination because of the presence of edema of the lower limbs which could not be accounted for by the condition of the heart or kidneys. His diagnosis and prognosis unfortunately proved to have been well founded In old subjects one should always look with suspicion upon such insidious edemas that cannot be shown to be either of cardiac or renal origin.

Much less commonly, one of the following types of edema may be encountered:

1. Edema of hepatic origin, nearly always of mechanical causation, *viz.*, pressure upon the inferior vena cava in advanced cirrhosis of the liver. Gilbert has, furthermore, described a *preascitic edema* met with early in the course of hepatic cirrhosis.

The supposedly established theory of an hepatic form of edema was seriously discredited by a few clinicians. In 1893 Hanot wrote: "Changes in the liver cells account for the cases of localized edema either about the malleoli or in the face in the absence of albuminuria. There exists an hepatic form of edema just as there is a renal edema and the former may be an early sign of disease." Le Damany, in a later contribution on the subject ("*Les hépatites hydropigènes*," 1914), concluded that there may be a defective elaboration of protein material in the liver, by virtue of which, such material being retained in the tissues, it may secondarily induce retention of water and chlorides Certain puzzling clinical combinations, *e.g*, pronounced edema with but little ascites, preascitic edema, and extreme ascites without edema, occur, indeed, which argue against the purely mechanical causation of edema of hepatic origin

2 Edema of nervous origin.—Edema may be witnessed in the presence of peripheral neuritis, of spinal affections such as tabes and syringomyelia, of hemiplegia, of epilepsy, of paralysis agitans, and of exophthalmic goiter Generally, however, if one carefully eliminates the edemas of cardiorenal origin which may occur in the presence of these disorders, one finds that, as a rule, the condition present is merely a dystrophic pseudo-edema in which the characteristic sign of pitting on pressure cannot be elicited

3. Dystrophic edema.—This is the so-called "*trophedema*" of *Henry Meige:* "The term *trophedema,* without further qualification, may be applied generally to the dystrophic edemas of as yet unknown cause, but seemingly of nervous origin.

"Chronic trophedema is characterized by a white, firm, painless edema affecting one or more segments of one or several limbs and persisting throughout life without notable prejudice to the health of the individual Sometimes the condition occurs

singly. In other instances it is an inherited and family disorder. It may also be congenital

"One may describe as acute trophedema the so-called neuropathic, circumscribed, angioneurotic, neurovascular, and intermittent edemas, etc.—transitory edematous involvements sometimes accompanied by thermic manifestations, by disturbances of sensation, by pain, by changed color of the skin, and frequently also by trophic skin disturbances, eruptions, or ulcerations. This group of conditions make up what is known as Quincke's disease" (Henry Meige)

**Special Localized Edema.**—Lastly, certain local forms of edema require mention, *viz*, *edema of the lower extremities*, *edema of the upper extremities*, and *edema of the face and lids.*

(*a*) **Puffiness of the face and lids.**

1. *Puffiness of the face* and slight edema of the eyelids are *normal* conditions upon awakening in some individuals. This is due to an unusual relaxed, flaccid state of the tissues in these persons. Such a quasi-normal edema is seen most frequently in women and obese subjects

2. *Puffiness of the face* is a well-known feature upon awakening *after alcoholic sprees.*

3. It is likewise of frequent occurrence in pregnancy, and seems to be one of the component features of the *facies of pregnancy.* As in the preceding forms, however, it is well none the less to test the urine for albumin.

4. It is met with in *eczema, erysipelas,* and *sunburn*

5. *Blepharo-conjunctivitis* is also attended with puffiness of the eyelids.

6. Edema of the surrounding tissues in *dental abscess* is characteristic.

7. *Furuncles in the nostrils* frequently induce a marked edema of the infrapalpebral or even the palpebral portions of the face.

8. *Tumors of the neck and mediastinum*—aortic aneurysm, posterior forms of pericarditis, Hodgkin's disease, Ludwig's angina, and much more exceptionally, thrombosis of the superior vena cava, cause edema of the face and neck, sometimes very widespread, extending at times to the upper portion of the thorax and to

the root of the upper extremities. Of all these causes, aortic aneurysm and pericarditis are the commonest.

9. The doughy facies of *myxedema* should likewise be borne in mind.

10. Certain *drug intoxications* (iodine, bromine, antipyrin) induce congestion of the mucous membranes with facial edema.

11 Finally, mention may be made of *angioneurotic edema* or Quincke's disease, characterized by the sudden appearance of edema of the extremities, accompanied by edema of the mucous membranes Often this condition is restricted to the lids (idiopathic edema of the lids).

(*b*) Edema of the upper extremities.—This is met with:

1. In *septic disorders involving the upper extremities* and the axilla.

2. In *phlebitis,* much less commonly, however, than in the lower limbs.

3. In *gout,* likewise much less commonly than in the lower limbs.

4. In *cervical and mediastinal pressure*

(*a*) Lymphatic enlargements, cervical and mediastinal.

(*b*) Malignant growths of the breast and mediastinum.

(*c*) Aortic aneurysm.

(*d*) Hodgkin's disease (mediastinal lymphoblastema).

5. In *malignant metastases* in the mediastinum or axilla.

It should be noted that:

1. Renal edema is rare in the upper extremities, and is seen only in a late stage, in the period of anasarca.

2. The same is true of edema of cardiac origin, which is only exceptionally observed in recumbent subjects who have been lying on one of their arms, causing venous obstruction and hindrance to the return circulation It should be carefully remembered that cardiac edema is a dependent or "gravity" edema

(*c*) Edema of the lower extremities —In contrast with the assertions made above concerning the upper extremities, the following kinds of edema nearly always appear first in the lower limbs·

1. *Edema of cardiac origin.*

2. *Edema of renal origin.*

singly In other instances it is an inherited and family disorder. It may also be congenital.

"One may describe as acute trophedema the so-called neuropathic, circumscribed, angioneurotic, neurovascular, and intermittent edemas, etc.—transitory edematous involvements sometimes accompanied by thermic manifestations, by disturbances of sensation, by pain, by changed color of the skin, and frequently also by trophic skin disturbances, eruptions, or ulcerations. This group of conditions make up what is known as Quincke's disease." (Henry Meige).

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## EPIGASTRIC PAIN.

[ἐπί, *over*; γαστήρ, *stomach.* *Over the stomach.*]

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The pit of the stomach or epigastrium, a region bounded above by the xiphoid appendix and the lower costal margins,

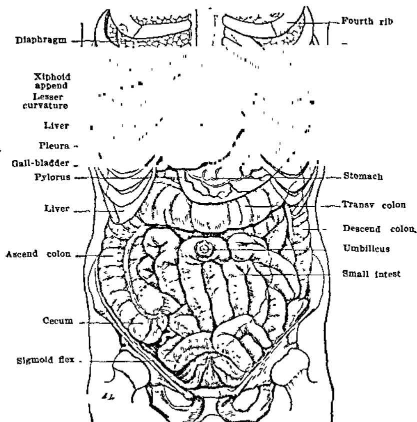



Fig. 612—General topographic anatomy of the abdomen (*Poirier*).

and below by a line passing midway between the umbilicus and the xiphoid, overlies, from before backward, the anterior aspect of the liver, the anterior surface, upper border, and posterior surface of the stomach, the pancreas, the celiac axis, and the

As in the case of the upper extremities, edema is here met with in the presence of.

1. *Septic affections of the lower extremities,* and particularly of the feet.

2. *Phlebitis,* relatively common in this region, particularly as puerperal phlebitis, phlegmasia alba dolens, post-operative phlebitis, etc.

Much commoner still is *varicose edema,* due to phlebosclerosis, almost always absent on arising from recumbency, but reaching its height before retiring, after having been in the vertical posture all day.

3 *Gout,* the seat of election of which, as is well known, is in the great toe.

4. *Abdomino-pelvic pressure*·

(*a*) Utero-ovarian tumors and cysts (cysts, fibromas, malignant or inflammatory swellings).

(*b*) Rectovesical malignant growths

(*c*) Primary or secondary enlargements of pelvic and abdominal lymphatics.

(*d*) Various abdominal tumors and cysts (hydatid cysts, various new growths)

5. *Malignant metastases*

6. Lastly, the lower limbs are the seat of election of the so-called *"cachectic" edemas,* of varying and complex causation.

The pathogenesis of edema and its significance in pathologic physiology have been the subject of very many investigations in the course of the last ten or fifteen years To present an outline of this subject, however important it is and tempting the occasion, does not come within the scope of this work. The reader desirous of obtaining an idea of some of the later studies on the causation of edema is referred to current medical periodicals.

Epigastric pain is generally an expression of some gastrohepatic disorder, such as gastrohepatic congestion, hyperchlorhydria and its attendant manifestations, gastric neurosis, ulcer or cancer of the stomach, or cholelithiasis of the gastralgic type.

Much more exceptionally it is an expression of disease of some adjoining organ, *e.g.*, pancreatitis, pericarditis, or abdominal aneurysm, or of disease of some remote structure, *e.g.*, appendicitis.

Among the possible causes mention should also be made of tabes dorsalis, with its frequently dramatic attacks of pain in

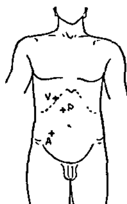



Fig 614—*A*, Surface projection of involved area in appendicitis, *P*, of involved area in pancreatitis, *V*, of involved area in cholecystitis.

the epigastrium (gastric crises), the pathogenesis of which is still rather obscure, and to which the term abdominal angina has been applied.

**Hepatic Syndromes**—The *pain of active or passive congestion of the liver is generally latent,* being elicited only by palpation or percussion It is most constant in the epigastrium in these cases. From the semeiologic standpoint, it is equivalent to *congestive tenderness of the liver.* Whenever it is met with, further investigation to ascertain its origin is indicated

1. Disorders of the biliary passages—cholelithiasis (hepatic colic, sensitiveness of the gall-bladder, jaundice, and the evidences of hyperchlorhydria).

solar plexus; still farther posteriorly is the lesser peritoneal cavity, and lastly, the aorta, which can often be seen or felt *pulsating in the epigastrium in thin individuals with atonic* musculatures.

Pain is frequently referred to the epigastrium, either as a

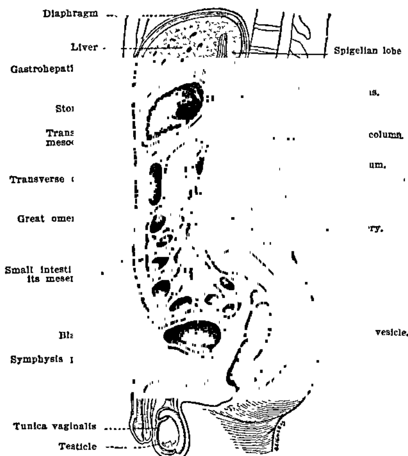



Fig 613—Sagittal section of the abdomen

dull, heavy sensation, especially after meals as in many dyspeptics—or as an acute and almost lancinating or even a piercing pain, coming on a few hours after or independently of the meals, as in gastric ulcer, the gastralgic form of calculous attacks, or the gastric crises of tabes—or brought on exclusively by pressure or percussion, as in many instances of congestion of the liver or gastric neuroses (hyperesthesia of the solar plexus).

*and gastric hyperacidity, generally in combination, are the main factors of the syndrome of hyperchlorhydria.*

These "theoretic" considerations have here been referred to at some length because they afford important therapeutic indications: Excessive peristalsis indicates administration of belladonna, and hyperacidity, of alkalies—both often remarkably efficient measures.

These considerations are completely applicable to the diagnosis and treatment of gastric ulcer. The symptoms of hyperchlorhydria, x-ray examination, and systematic testing of the stools for blood after the institution of a special diet are the main factors leading to a diagnosis, an obvious, outward demonstration of which may, however, in some cases be supplied in the form of actual hematemesis. In addition to the remedial measures already mentioned a protective "dressing" of bismuth in the stomach should be applied in these cases.

As for gastric neurosis with gastroptosis (dilatation of the stomach), gastrointestinal atony, frequently hyperacidity, in short, *hyposthenic dyspepsia,* the pain manifestations attending it are far different. These are dull pains coming on immediately after ingestion of food, with marked hyperesthesia of the solar plexus (permanent induced epigastralgia), sensations of weight or puffing, various local discomforts, vasomotor disturbances, etc.—all superimposed upon an asthenic and neuro- and psychopathic make-up which is frequently characteristic. It should be borne in mind, however, that occasionally, accidentally the pains may, in a few of these cases, assume the type met with in the "syndrome of hyperchlorhydria;" one of the surprises sometimes encountered in fluoroscopy is the observation of such a ptotic and habitually atonic stomach contracting with very great vigor at the time of evacuation. Observations of this sort are, in this connection at least, in favor of the hyperperistaltic theory of the causation of pain in these cases, the pain thus representing an actual "colic of the stomach."

The pain in cancer of the stomach is of very variable description, being sometimes almost *nil,* and only slightly elicited by palpation; at other times, a dull pain, with sensations of weight in the epigastrium and distention in the same region; more rarely it presents the delayed and "boring" attributes of the ulcer upon which the cancer has been superimposed (degenerated ulcer). Thus, in

2. Disorders of the liver—dyspeptic congestion due to excessive intake of alcoholic beverages and meats, precirrhotic hyperemia, abscesses, infectious jaundice, syphilis, or malaria.

3. Disorders of the heart in particular, passive congestion of the liver with tenderness being one of the most patent and constant signs of cardiac insufficiency (reduced function or actual heart failure).

**Gastric Syndromes.**—Pain thus caused exhibits very different features in two practically opposite conditions affecting the stomach, *viz.*, the *syndrome of hyperchlorhydria*, and *gastric neurosis with ptosis and gastrointestinal atony*. Indeed, the combinations of gastric symptoms are so protean that almost anything may occur, even an apparent coexistence of the two syndromes referred to.

Little space need be devoted to the well-known "hyperchlorhydric syndrome"—pain on gastric evacuation, "hunger pain"—the main feature of which is a combination of *tardy stomach pains*, sometimes quite severe and of the "burning," boring type, coming on periodically several hours after meals, and generally allayed by alkalies or bland foods, and a more or less pronounced hyperchlorhydria or even sometimes a gastroduodenal ulcer.

The pain in these cases had formerly been ascribed to irritation of the sensory nerves of the stomach, when exposed by an ulcer, by the unduly acid gastric contents As with any other careful clinical observation, the observed fact remains unimpeachable, and its interpretation alone has been subject to revision. The finding of the symptoms of "hyperchlorhydria" in persons with normal gastric acidity, physiological experimentation, and x-ray observations tend to show that gastric hypertonicity, pylorospasm, and exaggerated peristaltic movements play an important, if not predominant, rôle in the causation of this pain. That the oncoming of the pain is delayed is because it appears particularly during the period of evacuation of the stomach, consentaneously with especially forcible contractions of the pylorus There is thus likewise explained the observation of the syndrome referred to in many chronic disorders of the intestine and biliary passages causing excessive peristalsis in these structures. Examination of the very large aggregate of clinical and experimental data available leads, at any rate, to the conclusion that *hypertonicity of the stomach and intestines*

Doubt may persist even after operation. Twenty-five years ago the author had under observation, with the late Dr. Guinard, a case of chronic obstructive jaundice with marked loss of weight in a patient manifestly suffering from gall-stones; the operation showed a large stone blocking the bile-duct, which was removed, and also a scirrhous growth of the head of the pancreas. *De visu*, the diagnosis of cancer of the head of the pancreas was decided upon and the prognosis corresponding to this condition issued The patient not only recovered, but "bloomed out" again, and lived fifteen years longer. The condition present was an interstitial pancreatitis secondary to calculus in the bile-duct—a condition of which little or nothing was known at the time, and which Dieulafoy's work was instrumental in bringing to general notice.

2. Hyperacute epigastric pain, *with pancreaticoperitoneal hemorrhage.*—Dieulafoy's account of the *"pancreatic tragedy"* should be read over in this connection "Terrific and generally fatal symptoms suddenly develop at a time when little apprehension existed and in persons not exhibiting jaundice at the time. The patient is seized with violent pain in the umbilical region, in the epigastrium, and in the hypochondriac regions The highly distressing and excruciating pain is accompanied by vomiting, prostration, and a tendency to syncope; there is general abdominal hyperesthesia, constipation is absolute, and there is not the least passage of gas. In the presence of such a condition one thinks of an acute peritonitis, an attack of poisoning of some sort, or perforation of the stomach, duodenum, or gall-bladder, one thinks of appendicitis, or of intestinal obstruction, but none of these conditions exist, and I have applied to the attack the term 'pancreatic tragedy' in order to establish a clear distinction between it and all other conditions that may resemble it. At the operation or the autopsy, indeed, there is found, not a peritonitis, not a perforation of some organ, not an appendicitis, not an intestinal obstruction, but the major pathologic witnesses of the tragedy, *viz.*, the white foci (candle-grease spots) of fat necrosis, with which are frequently associated pancreaticoperitoneal hemorrhages—lesions consecutive to an attack of *acute pancreatitis*, nearly always superimposed, in turn, upon a *chronic pancreatitis*." The diagnosis, rarely correctly made, is based, on the whole, upon the history of pancreaticobiliary trouble,

these cases study of the pain may be misleading. The diagnosis should be based especially on the finding of blood in the feces (on a meat-free diet), the frequently pathognomonic results of fluoroscopic examination, the age of the patient, the degree of impairment of nutrition, and later on, the direct detection of a gastric tumor (see *Dyspepsia*).

**Cholelithiasis** *is frequently attended, as already pointed out,* with the syndrome of hyperchlorhydria, and differentiation from duodenal ulcer and hypersthenic dyspepsia is frequently a difficult task. A positive diagnosis in some instances may be reached only by careful examination for gall-bladder signs, such as gall-bladder pain and tenderness, radiation of the pain to the right shoulder, slight or distinct jaundice, definite attacks of hepatic colic, and a history of former infections (typhoid fever, etc.).

All of the preceding conditions giving rise to epigastralgia are very common The following are much less common, or indeed, strictly speaking, are exceptional:

The epigastralgia of **pericarditis** presents absolutely no distinctive feature; it is a dull, or even latent, pain, readily awakened by pressure. When one has had the opportunity to see and follow up cases of "already diagnosticated" pericarditis, the pain will regularly be thought of; in the opposite event, it will always be overlooked. Dyspnea, cardiac acceleration, and sometimes precordial pain will attract the observer's attention. Systematic examination of the case, including fluoroscopy, will afford a positive diagnosis.

The epigastric pain of **pancreatic origin** is met with under two *quite different clinical conditions:*

1. In the chronic form.—This is often the *pancreatico-biliary syndrome* of Dieulafoy (former gallstone disease, obstructive jaundice, loss of weight, pain, and sometimes a pancreatic mass in the right intercostoumbilical region). Yet, as Dieulafoy so correctly said, "even supposing that the maximum of pain is observed at the pancreatic point, that is, a point 4 or 5 centimeters to the right of, above, and laterally from the umbilicus (pancreaticocholedochian region), it is not always easy to find out whether the pain is to be attributed to pancreatitis or to stones in the bile-duct."

| CAUSES | KIND OF PAIN. | DURATION AND RECURRENCE | OTHER CLINICAL MANIFESTATIONS. | FEVER AND LEUCOCYTOSIS | CONDITION OF THE HEART. |
|---|---|---|---|---|---|
| Acute pancreatitis. | *Sudden and severe*, stabbing | A few hours. | Collapse; symptoms of peritonitis | + | 0 |
| Pericarditis. | *Dull, not* characteristic, induced especially by pressure | Weeks. | Dyspnea, increased pulse rate, signs of pericarditis with or without effusion. | + *or* 0 | Signs of the pericarditis. |
| Abdominal aneurysm | Pulsating, dull, deep-seated | Chronic. | *More or less forcible* pulsations of the abdominal aorta. | 0 | 0 |
| Appendicitis. | More or less acute, *coincidently with* pain upon pressure over the right iliac *fossa*. | Minutes or hours. | Nausea and vomiting. | + | 0 |
| Tabes (gastric crises). | Uncontrollable attacks of boring pain. | *A few minutes to a* few days. | *Attacks resembling* those of paroxysmal hyperchlorhydria + symptoms *and signs* of locomotor ataxia | 0 | 0 |
| *Angina abdominalis.* | *Anginose, "syncopal,"* with hyperesthesia of the solar plexus *and abdominal* and lumbar radiation. | A few minutes to a few hours. | Faintness or syncopal attacks induced by fatigue or pronounced emotion. Often associated with abdominal ptosis. | 0 | Frequently an associated cardio-arterial disorder (arteriosclerosis, hyposphyxia, etc ). |

| Causes | Kind of Pain. | Duration and Recurrence | Other Clinical Manifestations. | Fever and Leucocytosis | Condition of the Heart. |
|---|---|---|---|---|---|
| Gastrohepatic congestion (of varying origin, frequently cardiohepatic). | Dull; sensation of weight. Particularly induced by percussion | Continuous, for days and weeks. | Cardiac or hepatic disorder. | Occasionally. | Frequently mitral or mitroaortic disease in the stage of decompensation. |
| Syndrome of hyperchlorhydria. | Sharp, burning, sometimes almost "boring" | Late after meals Periodically when the stomach is empty. | Liver distended, pyrosis, acid regurgitations, sometimes vomiting, alkalies bring relief. | 0 | 0 |
| Gastric neurosis. | *Sensation of weight* and puffing, eructations, etc Cutaneous and deep-seated hyperesthesia. | *Chronic, with exacerbations.* | *Atony, asthenia, neuro*pathic disorder, impaired nutrition, and vasomotor disturbances | 0 | *Small Low blood*-pressure. |
| Gastric ulcer. | Boring, piercing, stabbing. | Periodically, when the stomach is empty. | Pyrosis, sometimes vomiting or even hematemesis Blood in the stools (Weber's test). | 0 | 0 |
| Gastric cancer. | Highly variable both as to type and intensity. | Chronic. | Impaired nutrition, sometimes *vomiting, hematemesis*, and mass in stomach Blood in the stools. | 0 | 0 |
| Cholelithiasis of gastralgic type. | Sudden and severe, of the gastralgic type, as in hyperchlorhydria. | A few hours. Repeated recurrence. | Tenderness of gall-bladder, sometimes jaundice | Occasionally. | 0 |

# EPISTAXIS.

[ἐπί, *upon*; στάζειν, *to drip*.
*Nasal hemorrhage.*]

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**Epistaxis** may be defined as a hemorrhage in the nasal cavities.

This symptom, when present, is obvious, and cannot be mistaken for any other unless, in posterior epistaxis—which,

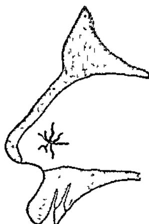


Fig 615—The artery of epistaxis Terminal distribution of the sphenopalatine artery. Upon raising the tip of the nose with the forefinger and inserting a speculum, the observer will notice in many persons, at the lower anterior portion of the septum, a small group of diverging arterial branches. If ulceration occurs at this point—usually on account of insertion of the finger—copious hemorrhage from one of these vessels will occur This is the source of the majority of cases of epistaxis (*Laurens*)

however, is uncommon—the blood, unwittingly swallowed, is later discharged by vomiting (suggesting hematemesis) or passes out with the stools (suggesting melena). The mere recollection of the possibility of these altogether exceptional mistakes is sufficient completely to guard against their occurrence; in the event of doubt, rhinoscopic examination, advantageous in any case, and often essential, will settle the question.

There are many *possible causes of epistaxis.*

the "tragic" syndrome above described, and particularly upon actual inspection of the lesions found upon laparotomy, which is indicated whatever condition may be thought present

**Aneurysm of the abdominal aorta** may be suspected—at least at first—only in the presence of the following combination of clinical indications: Pain in the epigastrium and diffuse, deep-seated pulsations in the same region. Careful fluoroscopic examination with the patient in an oblique position can alone afford a positive diagnosis.

**Appendicitis, whether acute or chronic,** may be attended with epigastric pain and even nausea and vomiting—epiphenomena of a syndrome of peritonitis or "peritonism" or hyperperistalsis, the frequency of which has already been referred to Only very exceptionally, however, will careful clinical examination not reveal the right iliac fossa as the probable source of the disturbance. For the required further discussion of these cases the reader is referred to the section on *Vomiting.*

Concerning **gastralgia in tabes** the same may be said as of epigastralgia in pericarditis, *viz.*, whoever has seen and followed one such case will always have the condition in mind, while whoever has not will never think of it. The onset is generally sudden, with extremely severe pain in all respects suggesting that of gastric ulcer or of hepatic colic, with unusual obstinacy, so that even morphine brings only partial relief; with vomiting, often uncontrollable; the duration of pain a few hours to a few days, and the cessation of pain sudden like the onset These features will put the well-posted observer on the right track Furthermore, **systematic examination of the patient (which should never be omitted)** will often discover a number of characteristic evidences, *viz.*, a history of syphilitic symptoms, loss of knee-jerks, Argyll-Robertson pupil, impaired equilibration, ataxia, etc. The attacks are often exactly similar as to onset, course, and duration, and the patient, having in mind an earlier diagnosis of his case, may even supply the diagnosis himself. It is well not to forget that tabetic gastralgia may be accompanied by gastric ulcerations—actual "perforating ulcers,"—which may result in hematemesis, sometimes very rebellious.

## EPISTAXIS.

[ἐπί, *upon*; στάζειν, *to drip*. *Nasal hemorrhage.*]

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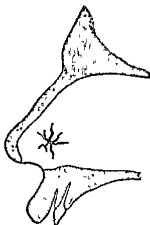


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There are many *possible causes of epistaxis*

**Locally**, epistaxis may be of *traumatic* (blow on the nose) or *operative origin* (the latter following, for example, removal of one of the turbinates, etc.); further reference to these obvious causes would be superfluous. In 90 per cent. of cases, *according to G.* Laurens, *epistaxis is due to a localized, varicose erosion of the anterior inferior portion of the nasal septum.* This is a clinical feature to be carefully remembered, since all local treatment (cautery, packing, pressure, etc.) is necessarily based on it.

**General causes** of epistaxis may be divided into 2 groups, *viz.*, the mechanical or circulatory and the dyscrasic or hemic. Like all other attempts at a simple, easily remembered classification, this division is in some respects questionable, most causes of epistaxis acting in a "mixed" manner, *i.e.*, both mechanically (through low blood-pressure and reduced resistance of the vascular walls) and dyscrasically (through hydremia, lowered viscosity, disturbances of coagulation of the blood, etc.), as in chronic nephritis or arteriosclerosis The division above made is merely of didactic value—which is the essential point herein

The **mechanical causes** comprise all factors resulting in congestion of the nasal mucosa, which is an exceedingly vascular and erectile tissue.

(*a*) **Passive, venous congestion of the nasal mucosa**, due to increased venous blood pressure in the superior vena cava. Under this heading belong mitral and tricuspid lesions with loss of compensation, heart failure in general, and pressure upon the veins of the neck or mediastinum; these are, on the whole, relatively uncommon causes of epistaxis, which are readily detected by virtue of the accompanying cyanosis, turgescence of the veins, dyspnea, frequent and irregular heart action, and even a cursory examination of the chest

(*b*) **Active, arterial hyperemia of the nasal mucosa**, due to high blood-pressure, is by far the most frequent cause of epistaxis. **All disorders attended with high blood-pressure**, including *plethora, gout,* and in a greater degree, *arteriosclerosis, aortic insufficiency,* and *Bright's disease* are, *par excellence,* conditions favoring epistaxis of the recurrent type and sometimes of alarming extent In the same group are, the epistaxis of *vicarious menstruation,* those of *hemorrhoids* and of the *menopause,* and in some degree, that

of *puberty*. Epistaxis is the most frequent type of hemorrhage witnessed in these conditions of high pressure with hemorrhagic tendencies, apparently because, most fortunately, the vessels of the nasal mucous membrane constitute a vascular *locus minoris resistentiae* or actual safety-valve affording, in the presence of dangerously high blood-pressure, a *"providential bleeding"* and a warning often of marked service when its significance is duly appreciated by the physician and patient. It may, however, in a given person occur in alternation with most varied types of hemorrhage, as in an arteriosclerotic patient with high blood-pressure, under the author's observation for over ten years, who developed every spring either epistaxis, hemoptysis, or hemorrhoidal bleeding and was finally rendered hemiplegic by cerebral hemorrhage In all these cases systematic blood-pressure estimations will supply the diagnosis as well as the prognosis, and bring out the various indications as to treatment. Systematic uranalysis, determination of the blood viscosity, and also, of course, general clinical examination, are essential in all instances.

The **dyscrasic causes or blood changes**, such as lowered coagulability, hydremia, etc, very frequently co-operate with the preceding causes in the production of the severe forms of epistaxis

As is well-known, certain blood disorders, including *anemias* and *leukemias*, frequently tend toward hemorrhage production. Cell counts, hemoglobin estimations, and the differential leucocytic count are the main diagnostic factors in these cases.

Other disorders, *viz.*, the *purpuras* and *hemophilic conditions* are as yet only rather imperfectly defined from the hematologic standpoint. Marked delay in coagulation is always noted These states are frequently related to more or less obvious deficiencies of the hepatic, renal, and polyglandular functions Indeed, clinically, the concept of inherited deficiency and the frequent, identical repetition of the hemorrhagic manifestations will compel the practitioner to make a provisional diagnosis of this nature—since these are clinical cases of a complex and probably dissimilar nature

Ever since remote ages there have been noted cases of *epistaxis symptomatic of various liver conditions* As a matter of fact this form of epistaxis is of very variable origin·

1. Dyscrasic infectious epistaxis in infectious "grave" icterus.

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## EXANTHEMATA. [ἔξω, *out of;* ἄνθος, *flower;* ἐξάνθεμα, *from* ἐξάνθειν, *to bloom. A skin eruption.*]

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Skin eruptions are of such frequent occurrence that it has seemed absolutely necessary to devote a semeiologic section to a brief presentation of the various types of eruption. These various types, however, by virtue of their actual frequency and number, do not lend themselves well to condensation into a short, yet comprehensive article. After prolonged hesitation the author decided to employ the following plan.

1. To give a didactic account of certain elementary facts in dermatology, based on the writings of an authority on the subject—Sabouraud

2 To recall, in a short synopsis, the main clinical features of the great chronic infectious skin disorder of temperate climates, *viz.*, syphilis

3 To recall, in condensed, *tabular form,* the more essential facts concerning the eruptive fevers.

4. To request the reader to refer to special works on dermatology for further details

### I ELEMENTARY AND ESSENTIAL FACTS IN DERMATOLOGY.

(AFTER SABOURAUD)[1]

There are some eruptive diseases that involve the entire surface of the body or the greater portion of it Such are the *exanthematic (eruptive) fevers*

On the other hand there are skin disorders which, without ever extending to involve the entire cutaneous surface, have no definite seat of election and may be noted in any portion of the body. Such are the *epitheliomas.*

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[1] See Sabouraud's *Dermatologie topographique,* p 581.

2. Dyscrasic non-infectious epistaxis of hepatic secretory insufficiency.

3. Dyscrasic and mechanical epistaxis in cirrhosis, particularly of the atrophic variety.

4. Mechanical epistaxis in congestion of the liver—either passive *congestion, as in increased venous pressure in the liver and cirrhosis* of cardiac origin, or active hyperemia, as in the increased arterial blood-pressure of plethoric or gouty enlargement of the liver, etc.

**Infections.**—Lastly, many *infections* are either especially and primarily producers of hemorrhage, or may occur in a hemorrhagic form. This applies particularly to·

1. **Typhoid fever.**—*Epistaxis* is, as is well-known, frequently a preliminary symptom in typhoid. In the *hemorrhagic form,* epistaxis may go hand in hand with hemorrhage from the bowel.

2. **Eruptive fevers,** particularly *measles,* chicken-pox, varioloid, and scarlet fever.

3. **"Rheumatism"** and "infectious pseudo-rheumatism" in some of their "purpuric" forms.

**EPISTAXIS.**

| | **Local Causes.** |
|---|---|
| 1. **Traumatism** . . . | (Obvious) |
| 2. **Operation** . . | (Obvious). |
| 3 **Inflammation** . | (Erosion of the anterior inferior portion of the septum; rhinoscopic examination) |
| | **General Causes.** |
| (a) **Passive venous congestion.** | 1 [illegible] |
| | 2 [illegible] cava, as by a tumor of the neck or mediastinum (cyanosis, sometimes edema, collateral circulation, and signs of tumor). |
| (b) **Active hyperemia (far more common).** | 1 All disorders attended with high blood-pressure· Plethora, gout, arteriosclerosis, Bright's disease |
| | 2. Certain physiologic states· Menopause and puberty |
| | 3 Vicarious menstruation or hemorrhoidal flux. |
| (c) **Dyscrasias.** | 1. Anemias, leukemias, purpura, and hemophilic states |
| | 2 Liver disturbances: Infectious jaundice, cirrhosis, or hepatic congestion |
| | 3. Infections promoting hemorrhage: Typhoid fever, the eruptive fevers, infectious rheumatism, etc. |

## EXANTHEMATA (*continued*).

| PRIMARY CHARACTERISTIC FEATURE | DERMATOLOGIC APPELLATION | SEMEIOLOGY. |
|---|---|---|
| 6. **Pustules,** or purulent vesicles. Ruptured pustules become more or less superficial ulcerations. | Pustular and ulcerative skin affections. | Staphylococcic pustules.<br>Furuncle. Carbuncle<br>Acne pustules<br>Phlyctenosis streptogenes.<br>Ecthyma.<br>Small-pox Varioloid. Chicken-pox |
| 7. **Bullæ,** or large vesicles, "balloon-like" and constituting the so-called pemphigoid eruptions. | Bullous and pemphigoid eruptions. | Urticaria bullosa.<br>Erythema multiforme bullosa<br>Bullous drug eruptions<br>Acute infectious pemphigus<br>Various forms of pemphigus (vegetating, foliaceous, traumatic, or hysterical)<br>Painful, multiform dermatitis (Duhring, Brocq) |
| 8 **Purpura,** consisting of blood macules ecchymotic spots, or extravasations into the skin which pressure with the finger cannot dissipate | Purpura and purpuric eruptions. | Hemophilia.<br>Rheumatic purpura (peliosis rheumatica)<br>Werlhoff's disease<br>Acute febrile purpura.<br>Cachectic purpura<br>Toxic purpura<br>Purpuric eruptions in eruptive fevers (exanthemata with purpura) |
| 9. **Erythematous macules,** hyperemic spots temporarily dissipated by pressure with the finger. | Measles and rubeoliform eruptions | Measles<br>Rubella<br>Febrile roseolæ (typhoid fever, small-pox, puerperal fever, etc )<br>Syphilitic roseolæ<br>Drug roseolæ<br>Roseola due to serum disease. |
| [illegible] | [illegible] | Scarlet fever<br>Scarlatinoid rash in small-pox.<br>Recurrent exfoliative scarlatinoid erythema.<br>Toxic scarlatinoid erythemas.<br>Mercurial rash |
| 11 Erythrodermias consisting mainly of erythematous lesions with edematous infiltration of the dermal layer. | Erysipelas and erythrodermias. | Febrile erysipelas<br>Exfoliative erythrodermias.<br>1 Generalized, afebrile, and primary.<br>2 Secondary (traumatic, mercurial, and arsenical eruptions) |

## EXANTHEMATA.

| Primary Characteristic Feature | Dermatologic Appellation | Semeiology. |
|---|---|---|
| 1. Some skin disorders are characterized solely by dry scales and exfoliation of the horny layer, without redness or oozing of fluid. | **Simple squamous skin affections.** | Ichthyosis (congenital).<br>Desquamation in febrile states and eruptive fevers (scarlet fever).<br>Pityriasis in various forms:<br>— simplex.<br>— (tinea) versicolor (mycotic)<br>— rosea of Gibert.<br>— rubra pilaris.<br>Dry eczema<br>Psoriasis |
| 2 Flat elevations, with itching, exactly resembling nettle rash | **Urticarial affections.** | Nettle rash<br>Dermographism (vasomotor neurosis).<br>Toxic urticarias.<br>Food poisoning<br>Drug poisoning<br>Idiopathic urticarias of unknown cause |
| 3. Parasitic lesions, lesions due to scratching, attended with itching and easily confused with prurigo | **Parasitic skin affections.** | See Pruritus.<br>Scabies Pediculosis. Fleas<br>Bedbugs Mosquitoes |
| 4 [illegible] patches. | [illegible] | Papular syphilides<br>Lichen planus<br>Papulonecrotic tuberculides<br>Prurigo in various forms:<br>symptomatic;<br>senile;<br>diathetic, and<br>regional or circumscribed |
| 5 Vesicles, small, clear collections of fluid slightly raising the superficial epidermis; like "small pearls" embedded in the epidermal layer When broken, the vesicles discharge fluid. | **Vesicular and exudative skin affections.** | Eczema (initial eczematous vesicle, patch of eczema, with itching, oozing, crust formation, desiccation, and lichenification)<br>Miliaria sudoralis.<br>Chicken-pox.<br>Vesicular urticaria<br>Diffuse impetigo<br>Pemphigus foliaceus. |

Again, there are some which have elective localizations, yet spread over the entire body surface and therefore require a general description. Such is *scabies*

These disorders are in themselves very many, and necessitate a classification for the practitioner. Such a classification should be planned on lines sufficiently simple so that the physician will be able, without any previous dermatologic study, to ascertain easily the dermatologic type to which any disorder under observation belongs.

Condensed in the tables presented on the succeeding pages will be found:

**The main symptomatic features and course of syphilis.**

**The main symptomatic features and course of the eruptive fevers.**

* * *

The following statistical matter, collected by Halperin at Camp Lee, Petersburg, Va, gives a good idea of the approximate and respective frequency of the various skin disorders in young recruits upon their arrival in camp. Among 8000 subjects examined during the week following their incorporation Halperin found.

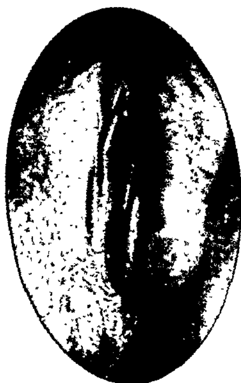
SKIN CONDITIONS IN 8000 RECRUITS

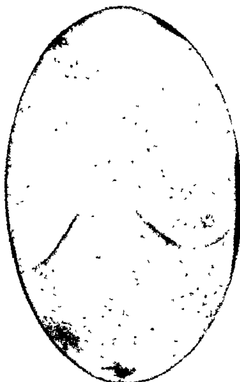
| Condition | Number | Condition | Number |
|---|---|---|---|
| Syphilis (open lesions) | 125 | Brought forward | 443 |
| Tinea versicolor | 109 | Sebaceous cysts | 3 |
| Pustular acne | 75 | Ringworm | 3 |
| Psoriasis | 40 | Erythrasma | 2 |
| Scabies | 20 | Herpes zoster | 2 |
| Seborrheic eczema | 18 | Varicose ulcers | 2 |
| Chronic eczema | 16 | Purpura | 2 |
| Folliculitis | 11 | Pediculosis pubis | 3 |
| Alopecia areata | 8 | Pediculosis corporis | 1 |
| Ichthyosis | 8 | Parapsoriasis | 1 |
| Urticaria | 5 | Lupus vulgaris | 1 |
| Erythema multiforme | 4 | Acne rosacea | 1 |
| Lipoma | 4 | Keloid acne | 1 |
| | 443 | | 465 |

Again, the war resulted in a renaissance of artificial or simulated eruptions. Such eruptions may be of an eczematoid aspect, or pustular, or pemphigoid, or may suggest mucous patches, sloughs, etc.

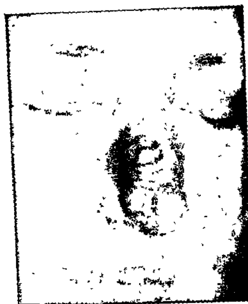
## EXANTHEMATA (*continued*).

| Primary Characteristic Feature | Dermatologic Appellation | Semeiology. |
|---|---|---|
| 12 Dyschromia, melanodermia, vitiligo, scleroderma, and morphea. | Dyschromia. Scleroderma. | Symptomatic dyschromias, as in albinism, pigmented nevi, lentigo, ephelides, xeroderma pigmentosum, and neurofibromatosis.<br>*Nervous dyschromias*, as in Addison's disease, tuberculous *melanodermia*, pigmented syphilis, leprous discolorations, and vitiligo<br>*Hematic dyschromias*, as in lymphadenosis, leukemias, mycosis fungoides, malarial cachexia, and bronzed diabetes<br>Toxic dyschromias, as in poisoning by arsenic, antipyrin, silver, or lead.<br>Dyschromias of local causation, as by heat, counterirritation, or pediculosis<br>Scleroderma |
| 13 Tumors of the skin, cutaneous neoplasms. | Skin tumors. | Retention cysts and the like benign tumors. Milium, sebaceous cysts, wens, hygromas, etc.<br>*Small, transmissible benign* tumors· Molluscum contagiosum, warts, and papillomas<br>Parasitic tumors: Blastomycosis, bothriomycosis, and keloids.<br>Circumscribed congenital skin deformities or nevi. Pigmented nevi, vascular nevi, and verrucose or fibromatous lymphangiomas.<br>*Dermatomyomas*<br>Dermoid cysts.<br>Fibromas<br>Lipomas<br>Xanthomas<br>Sarcomas<br>Mycosis fungoides, cutaneous lymphadenosis; papillary or *pearl-like epitheliomas, rodent ulcer*, etc. |


I


II


III


IV

EXPLANATION OF THE ANNEXED PLATE.

Typical Manifestations of Syphilis in its Several Stages.

1. *A primary chancre of the vulva,* fully developed The appearance is quite characteristic If it be further recollected that the chancre is indurated and accompanied by multiple, painless, inguinal glandular enlargement, with the central node particularly enlarged, an accurate mental conception of the ordinary syphilitic chancre and its accompaniments will be had. (PHOTO BY DR RAVAUT)

2 *A florid roseola* of the "peach-bloom" variety, distinguished from the non-specific "roseolar" eruptions only by the history of a chancre and the subsequent secondary manifestations on the skin and mucous membranes, together with the Wassermann reaction, which is always positive in this stage (PHOTO BY DR RAVAUT)

3 *Hypertrophied mucous patches of the vulva,* which, when so typical as those presented, could hardly be confounded with any other condition involving the genitals One should get accustomed to looking for smaller and more discrete patches than these (PHOTO BY DR. BROCQ).

4 *Ulcerated syphilomas of the nose,* which increasingly accurate diagnosis and energetic treatment are rendering daily more uncommon Note the regular, "cyclic" appearance of the ulcers, with their indurated margins and necrotic bases, these lesions are attended with surprisingly little pain The most striking advantage of arsphenamine and mixed (iodide and mercury) treatments is that they will heal and "dissipate" such ulcers as these within a few days (PHOTO BY DR. BROCQ)

*The author's thanks are due to Drs P Teissier, Brocq, and Ravaut, who kindly consented to select from among their valuable collections some excellent autochrome reproductions of skin conditions, thus enabling him to place before his readers a few of the types of cutaneous disturbance most commonly met with*

The diagnosis in these cases of malingering is always difficult, the patient obstinately refusing to confess his own responsibility in the matter. As in other forms of malingering, the suspicious appearance of the patient, the unusual sort of eruption, the lack of harmony between different symptoms and signs, the duration of the disease and its unaccountable recurrence, in short, the incoördinate or, on the other hand, repeatedly coordinate course of the disorder will eventually lead to a presumption and probable diagnosis of the condition.

* * *

On succeeding pages will be found a few chromo-typographic typical illustrations of the chief infectious eruptive disorders These will assist in making one familiar with the characteristic eruptions of rubella, measles, chicken-pox, small-pox, and scarlet fever, and *ipso facto* with the typical features of vesicular (chicken-pox), morbilliform (rubella and measles), scarlatinoid (scarlet fever), and pustular (small-pox) skin disorders.

## GENERAL REMARKS CONCERNING THE DIAGNOSIS OF SYPHILIS.

Syphilis, tuberculosis, and alcoholism are unquestionably three of the greatest scourges with which the human race is compelled to contend. No more imperative task devolves upon the practitioner than that of detecting them in whatever form they may appear.

As for syphilis in particular, such endeavor is all the more necessary in that: 1. The earlier the diagnosis is made, the greater the chances of radically effective treatment. 2. We are possessed of antisyphilitic remedies which are extremely powerful if administered early and with due energy and wisdom

The succeeding tables, together with the annexed illustrations, will give a general idea of the clinical manifestations of syphilis and, properly comprehended and availed of, will greatly facilitate its diagnosis. Let it be particularly borne in mind that the skin and mucous membrane eruptions of syphilis are merely the outward manifestations of a deep-seated septicemia; that the entire system is impregnated with the syphilitic virus, and

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that especially the cardiovascular and nervous systems are always, or nearly always, more or less attacked by the infection. Hence, let it not be thought that a syphilitic has been cured simply because he has been freed of all manifestations of the disease affecting the skin and mucous membranes; and let it not be asserted that an individual never had syphilis before because he has retained no obvious stigmata of the disease.

The diagnosis of syphilis, to-day as in the past, remains, as was happily stated by Fournier, one of a series of morbid states based on a definite diagnostic triad, *viz.*, the history, the existing signs of syphilis (in the skin, mucous membranes, and internal organs), and a practically specific serum test (the Wassermann reaction). This statement must be adhered to with precision, judicious consideration, and common sense, if the practitioner is to avoid making one of two almost equally fearsome mistakes, *viz., either of overlooking syphilitic infection which is still* a source of danger, or of burdening an individual free of syphilis with an obsessive and depressing idea that he is syphilitic. Few problems in diagnosis bring so actively into play the moral responsibility of the practitioner.

The three classic laws relating to congenital syphilis may here be recalled

**Law of Colles** (1837): A child born syphilitic of a syphilitic father generally does not infect the mother if she shows no signs of the disease, and she may nurse it without risk. To this we may add, with Carle (of Lyons): But she should be placed under treatment at once, as she harbors specific infection.

**Law of Profeta** (1865) The healthy child of a syphilitic mother cannot contract syphilis during lactation nor through any form of contact To this we may add, with Carle: But the child should be kept under observation, and even given treatment if indicated, as he is often syphilitic himself. Most cases of late inherited syphilis are merely exhibiting delayed symptoms of an unrecognized congenital syphilitic infection.

**Law of conceptional syphilis** (Diday; Fournier): A syphilitic fetus in utero may infect its mother through the placental vessels, with resulting appearance of secondary symptoms in the pregnant mother without any primary stage.

## MAIN SYMPTOMATIC FEATURES AND COURSE OF SYPHILIS.

**Primary stage** (*Average duration 2 to 3 months*) —POSITIVE WASSERMANN CONSTANT (100 PER CENT ) IN ANY CASE OF SYPHILIS OF MORE THAN TWO WEEKS' STANDING.

1 **Initial lesion or chancre** (see Genital Ulcerations), 15 to 20 days after suspicious coitus; ulcer generally single, without oozing, with board-like, cartilaginous induration; 5 to 10 millimeters in diameter; spontaneous recovery in 4 to 6 weeks (spirochetes in serum from the chancre)

2 **Lymphatic enlargement,** corresponding to the seat of the chancre, painless, indurated, larger than a filbert, coexisting with the chancre, and persisting 5 or 6 months longer, without *suppuration*

3 **Subsidiary lymphatic enlargement** of nodes surrounding the preceding, with the same features, but with the nodes smaller and multiple, no suppuration

**Secondary stage** (*Average duration 18 months to 2 years*) —POSITIVE WASSERMANN (IN 95 PER CENT ).

1 **General lymphatic enlargement,** involving successively most of the superficial groups of lymph-nodes and probably the deep groups, one should examine particularly the cervical, inguinal, axillary, and epitrochlear nodes; duration, 10 to 15 months

2. **Roseola** (secondary eruption) Occurring 70 to 80 days after infection, or 50 to 60 after the chancre, confluent or discrete spots or macules, situated mainly on the trunk and limbs, duration 2 weeks to 3 months Sometimes a "florid" eruption with fever and general discomfort, at other times so discrete that it may be overlooked

3 **Frequent pains, especially headache** (secondary cephalalgia recurring at night) and osseous, osteocopic pains, particularly in the tibias

4 **Secondary papules,** always occurring later than the roseola, round, more or less confluent, red brown or copper-colored papules, not changing into ulcers, duration 4 to 6 weeks.

5. **Mucous patches,** met with on all mucous membranes (mouth, genitals, anus); red, oval, ulcerations with a gray areola; symptoms of sore throat, laryngitis, etc , infectious lesions (spirochetes in the discharges).

6 **Alopecia, patchy,** of the scalp and eyebrows More rarely, iritis. Exceptionally onychia.

## MAIN SYMPTOMATIC FEATURES AND COURSE OF SYPHILIS (*continued*).

**Tertiary stage** (*Average duration Many years*) —POSITIVE WASSERMANN (IN ABOUT 75 PER CENT.).

*May be wanting; is generally separated from the secondary stage by a variable period of latency.*

1. Superficial skin lesions **Papular or papulotubercular** or papulogummatous syphilides, of regional distribution, limited, localized, grouped in bunches, corymbs, or circles, if ulceration occurs, there result serpiginous, tertiary, ulcerative syphilides
2. Deep-seated visceral lesions **Deep-seated syphilomas or neoplastic gummas** terminating in necrosis or fibrosis, **fibrous degeneration of organs;** these may involve any organ, more particularly the tongue, liver, kidneys, bones (nasal framework, tibia, etc ), genital organs (testicles), aorta (aortitis and dilatation), the vessels (particularly cerebral arteritis and perivascular fibrosis), nervous system (tabes dorsalis, general paralysis, and progressive muscular atrophy).

Some of these lesions, especially those affecting the nervous system (tabes and general paralysis), have for some time been termed parasyphilitic (Fournier), to recall their obvious relation to syphilis as well as the customary failure of antisyphilitic treatment. In this respect, many cases of aortitis and aortic dilatation manifestly of syphilitic origin *present precisely the same features* There does not seem to be sufficient reason for separating them from the other tertiary syphilitic manifestations Their incurability is probably due to the fact that at the time when the diagnosis is made the affected structures are already the seat of fibrous degeneration, a condition as yet refractory to our therapeutic resources, and that the patient has already passed *from the stage of disease to that of* infirmity (which does not mean, however, that one is completely helpless against this type of disturbance).

### Congenital Syphilis.

**Frequency of abortion of syphilitic origin, induced** { either by fetal syphilis (miscarriage from the 5th to the 9th month). or by embryonal syphilis (abortion before the 5th month).

Congenital syphilis proper may be roughly divided into.

**Early inherited syphilis,** already present at birth or appearing before the 6th month, and manifested in:

1. Skin lesions (exceptionally a roseola; papular, erythemato-papular, erosive, and gummatous syphilides, etc ) in all respects similar to those of acquired syphilis, sometimes pemphigus (syphilis bullosa), chiefly upon the hands and feet, present at birth, and both specific and characteristic.
2. *Lesions of mucous membranes;* the most important are syphilitic coryza, labial fissures, and fissures at the conjunctival commissures.

3 Bony lesions of the skull, ulcerative and fungous in type (together with the square-shaped head, boat-shaped or natiform skull, microcephalus, hydrocephalus), also of the long bones: periostoses, exostoses, gummas, dactylitis (syphilitic spina ventosa), syphilitic pseudo-paralysis of the newborn.

4 Visceral lesions *Digestive* disturbances (vomiting, diarrhea), enlarged liver and spleen, syphilitic orchitis, pathologic changes in the special sense organs (keratitis, *iritis*, *choroiditis*, retinitis, otitis, etc )

5 Impaired general health—an inconstant feature—which may pass into actual cachexia

If the patient survives, he may present a robust and practically normal appearance, or, on the other hand, exhibit the stigmata of

Late inherited syphilis, appearing from childhood up to adult life and manifested in

**1.** Skin lesions Tubercular or tuberculo-ulcerative syphilides and gummas of the skin.

**2.** Lesions of the bones and joints (*a*) Osteoperiostitis, especially involving the skull and tibia and terminating in large exostoses and deformities (saber tibia, flattened or upturned nose, perforation of the soft palate).

(*b*) Joint involvement, ranging from hydarthrosis to syphilitic pseudo-white swelling

3 Visceral lesions identical with those of acquired syphilis

4 Special sense lesions, more particularly of the eyes (keratitis, iritis, more rarely, retinitis, choroiditis, etc.) and the ears (deafness, specific *otitis*)

5 Dystrophic dental lesions (microdontia, unusual susceptibility to caries, erosions, *malformations*, Hutchinson's teeth, etc.).

(The ocular lesions, aural disturbances, and deformities of the teeth constitute Hutchinson's well-known diagnostic triad)

Furthermore, the **most constant feature,** the most definite stigma of inherited syphilis is **disturbed nutrition (dystrophy):**

Disturbed nutrition of individual structures, particularly the bones, teeth, special sense organs, testicles, breasts, and nervous system

General disturbance of nutrition, ranging from simple debility to congenital malformations, dwarfism, and infantilism

**Any congenital disturbance of nutrition should awaken a suspicion of inherited syphilis.**

## MAIN SYMPTOMATIC FEATURES AND COURSE OF SYPHILIS (*continued*).

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2. Lesions of mucous membranes; the most important are syphilitic coryza, labial fissures, and fissures at the conjunctival commissures.

| | | | | | | |
|---|---|---|---|---|---|---|
| 8 to 10 days. | 3 to 5 days | itis, photophobia, seromucous coryza, catarrh of larynx with roughened voice, and sometimes *stridulous breathing.*<br>(b) Enanthem in mouth and pharynx; *small red elevations* on the palate, erythematopultaceous stomatitis, with swelling and redness of the mucous membrane of the cheeks and *gums*<br>**Koplik's Sign.** Red spots, with a bluish point at the center, of pinhead size, situated on the inner surfaces of the cheeks and *lips*<br>(c) **Temperature** between 100.4° and 103.1°, with morning remissions and frequently a drop on the day before the eruption appears | 5 to 8 days | Small pink or bright red spots, *slightly* raised, soft and velvety to the touch, sometimes definitely papular, rounded or oval, some discrete, *others* grouped in semicircular figures, with intervening areas of normal skin.<br>The catarrhal *manifestations in the* nose, eyes, and larynx increase in intensity. Bronchial catarrh.<br>Temperature rises again to *102.2°* or 104° or even higher, then subsides along with the eruption | the eruption disappears and lasting a few days. | 16 days. |

## MAIN SYMPTOMATIC FEATURES AND COURSE OF THE ERUPTIVE FEVERS.

| Incubation Period | Stage of Invasion. | | Eruption | | Desquamation | Legal Period of Isolation. |
|---|---|---|---|---|---|---|
| | Duration. | Chief Symptoms | Duration | Main Features | | |
| | | | **Scarlet Fever.** | | | |
| 4 or 5 days | A few hours to *48 hours.* | Onset abrupt with. (a) Fever (104° or over). (b) Frequent pulse (140 or more) (c) Vomiting (d) Sore throat. Diffuse redness of soft palate, faucial pillars, tonsils, and pharynx, sometimes *swelling of the tonsils* and whitish exudates Enlargement of submaxillary glands. | 5 to 7 days or a few hours or 10 to 15 days. | First appears on the neck, chest, abdomen, and inguinal regions, extends to the arms and lower extremities, often spares the face Diffuse redness covering large skin surfaces without intervening normal areas; minute papular elevations of a more intense red color imparting to the palpating finger an impression of slight roughening Color ranging from pink to dark red, particularly marked at folds of the skin Strawberry tongue Temperature Above 104°; later recedes as the eruption fades | **Does not begin** before the 10th day Slight from the neck, then as fine scales from the trunk and extremities. Large flakes from the extremities, like glove fingers. To be looked for *especially on the* palms and soles | 30 to 35 days |
| | | | **Measles.** | | | |
| | | (a) **Catarrhal** symptoms affecting eyes and nose, with lachrymation, slight conjunctiv- | | Appears first on the face; then spreads to the neck, trunk, and *upper limbs (2d day); then to the* abdomen and lower limbs (3d day). | Desquamation very slight, *furfuraceous,* beginning rapidly as soon as | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 8 to 10 days | 3 to 5 days | ιobια,<br>cor-<br>ℎ of<br>ugh-<br>and<br>ιdu-<br>:.<br>ι m<br>ɔhar-<br>l ele-<br>pal-<br>opul<br>ιtitis,<br>and<br>ɾnu-<br>e of<br>and<br>Red<br>blu-<br>the<br>head<br>1 on<br>faces<br>and<br>e be-<br>and<br>ιorn-<br>and<br>drop<br>ᐧfore<br>p- | 5 to 8 *days* | Small pink or *bright red spots*, slightly raised, soft and velvety to the touch, sometimes definitely papular, rounded or oval, some discrete, others grouped in semi-circular figures, *with intervening* areas of normal skin<br>The catarrhal manifestations in the nose, eyes, and larynx increase in intensity Bronchial catarrh.<br>Temperature rises again *to 102.2°* or 104° or even higher, then sub-*sides* along with the eruption | the eruption disappears and lasting a few days. | 16 days. |

MAIN SYMPTOMATIC FEATURES AND COURSE OF THE ERUPTIVE FEVERS (*continued*).

| Incubation Period. | Stage of Invasion. | | Eruption | | Desquamation. | Legal Period of Isolation. |
|---|---|---|---|---|---|---|
| | Duration. | Chief Symptoms. | Duration | Main Features | | |
| | | | | **Rubella.** | | |
| 12 to 14 days. | A few hours. | Sometimes no symptoms, the eruption being the initial event<br>*Occasionally* a macular rose-colored enanthem on the palate<br>Absence of catarrhal symptoms and stomatitis.<br>*Temperature:* Normal or 100.4° to 101.3°. | 3 to 4 days. | Sometimes *spreads from above downwards*, beginning with the face.<br>Sometimes begins on the trunk<br>Sometimes appears simultaneously on several parts of the body.<br>May exhibit several forms:<br>(a) Morbilliform—(b) Scarlatinoid<br>(c) Polymorphous. Morbilliform at certain points and scarlatinoid elsewhere<br>*Suboccipital and cervical glandular* enlargement.<br>Fever: Trifling or evanescent. | Very slight, furfuraceous, sometimes wanting | 16 days. |
| | | | | **Small-pox.** | | |
| 10 to 12 days. | 2 to 4 days | Onset marked by:<br>(a) One or more chills, with diffuse pains and sweats.<br>(b) Headache.<br>(c) Backache.<br>(d) Vomiting. Diarrhea<br>(e) Temperature: 104° to 105.8° for | 8 to 10 days | (a) Enanthem, often preceding the exanthem, seen on the mucous membranes of the mouth, palate, and pharynx, pustules soon followed by ulcers; salivation; dysphagia; enanthem on the larynx, causing aphonia, or on the conjunctivæ or nasal *mucosa.*<br>(b) Exanthem, beginning on the face and *becoming general in 36* hours | Desiccation of the lesions about the 12th or 14th day.<br>The crusts fall off on the 15th to the 20th day *from the face,* later from the trunk and extremities (15 | Until complete recovery. |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | 2 to 3 days, then a variable *remission* on the day before the eruption appears.<br>(f) Prodromal rash toward the close of the period of invasion, inconstant; morbilliform or scarlatinoid or purpuric, developing over the groins, thighs, abdomen, and axillæ<br>Delirium, sometimes convulsions. | | 1st day. Red rounded macules, which become raised into domelike or blunt-pointed elevations (papules).<br>2d day. Vesicles filled with clear fluid.<br>3d day: The fluid in the vesicle becomes turbid and there is *formed an umbilicated* pustule surrounded by a red areola On the face, the pustule is surrounded by a zone of edema, especially on the eyelids<br>(c) **Fever:** Rises again with the stage of suppuration | days to one month), leaving *pale, irregular,* and sometimes permanent scars | |
| **Chicken-pox.** | | | | | | |
| **14 to 16 days.** | 1 to 2 days. | (a) Usually the erup*tion is the initial* sign of the disease<br>(b) Where the onset is severe, smallpox may be suspected (temperature 102 2° to 104°)<br>Pain in limbs Headache Backache Vomiting More rarely, convulsions The constitutional symptoms lessen, | **8** days. | (a) **Enanthem**, sometimes preceding *the exanthem; vesicles on the* palate, mouth, and pharynx, soon replaced by erosions<br>(b) **Exanthem.** May appear first either on the head, trunk, or extremities, begins with red or pinkish macules which very soon develop apical vesicles or bullæ *filled with clear fluid, ranging in* size from millet seeds to small grapes<br>By the second day the fluid in the vesicles becomes turbid, and the | Rapid desiccation, already manifest by the second day of the eruption The crusts fall off after **7 or 8** days. | 16 days. |

## MAIN SYMPTOMATIC FEATURES AND COURSE OF THE ERUPTIVE FEVERS (*continued*).

| Incubation Period | Stage of Invasion. | | Eruption. | | Desquamation. | Legal Period of Isolation. |
|---|---|---|---|---|---|---|
| | Duration | Chief Symptoms. | Duration | Main Features | | |
| | | | | **Chicken-pox** (*continued*) | | |
| | | *however, as soon as* the eruption appears.<br>Very exceptionally: A morbilliform or scarlatinoid rash. | | *vesicles become pustules*, which rapidly dry up. Rarely umbilicated bullæ are observed.<br>Lesions widely scattered, separated by large areas of normal skin.<br>Successive crops, each accompanied by a recrudescence of fever, macules, vesicles, and pustules, still moist or dried, may thus be seen at the same examination.<br>No stage of suppuration nor suppurative fever. | | |
| | | | | **Vaccinia.** | | |
| 3 to 4 days | | No stage of invasion. | 10 to 13 days. | By the 3d or 4th day there appears a small, *red, raised papule*.<br>On the 5th day the papule turns into a vesicle and becomes surrounded by a red zone.<br>On the 6th day there is a small pustule, which thereupon broadens out and becomes umbilicated.<br>On the 7th and 8th days the center consists of a yellow, umbilicated *crust, surrounded by a drop of* dull white or pearly puriform fluid; all around it is a bright red zone of hyperemia. | From the 10th *to the 13th day*, desiccation occurs.<br>From the 13th to the 20th day, the scab loosens and drops off, leaving an irregular, *permanent scar*. | |

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| **Erysipelas of the Newborn.** | | | | | | |
| 3 to 4 *days* after birth, occasionally from the 10th to the 17th day | | **Onset** sudden, with slight rise of temperature, depression, and crying. | **8** days. | Upon removing the infant's clothing there is seen a red patch beginning at the stump of the cord, then spreading to the scrotum or vulva, *the lower* extremities, and finally upward along the trunk.<br>Skin bright red; subjacent cellular tissue exhibits a brawny induration, a raised margin surrounds the affected area<br>Temperature 104°.<br>*Death in collapse* about the 8th day. | | |
| **Ordinary Erysipelas.** | | | | | | |
| A few days | A few hours, rarely **1 or 2** days. | **Onset** a few days after abrasion of the skin of the face or elsewhere, after vaccination, circumcision, etc , constitutional symptoms appear<br>(a) A single chill<br>(b) Vomiting<br>(c) Rather often a slight sore throat.<br>(d) Temperature 104° | **6 to 10** days | **Onset** in the face or at *some point* of the body near a wound<br>Red, hard, tender area, with elevated margins, gradually advancing to new tissue<br>Temperature· 103 1° to 104°. | Fine, scaly desquamation. | |

EXPLANATION OF THE ADJOINING PLATE.

The commoner types of infectious exanthemata.

1 Chicken-pox (varicella), the type of the vesicular exanthemata.

2 Small-pox (variola), the type of the pustular exanthemata, illustrating clearly the essential eruptive lesion (the pustule).

3 Scarlet fever (scarlatina), the type of the scarlatinoid exanthemata, affording a good illustration of the maximal degree of eruption at the natural skin folds

4 Rubella (German measles), a rare, seasonal, epidemic, contagious disorder characterized "by a general glandular enlargement, itching, and a rash" (Sabouraud).

*5. Measles (rubeola), the type of the "morbilliform" rashes.*

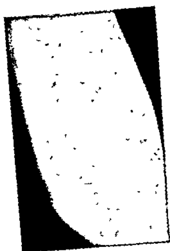
6. Florid measles, almost purpuric in appearance, constituting, from the eruptive standpoint alone, a manifest transitional form between the morbilliform rash (hyperemic) and the purpuric eruption (hemorrhagic).

*The author is indebted to Prof Pierre Teissier for the six original* pictures comprised in this plate.

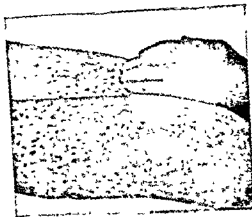

I


II


III


IV


V


VI

## EXPECTORATION. [εξ, *out of*, pectus, *chest; expulsion of abnormal secretions from the respiratory tract.*]

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Expectoration is, together with *cough*, the commonest manifestation of affections of the respiratory tract. While sometimes devoid of any great diagnostic importance, under certain other circumstances it assumes an exceedingly characteristic significance, as in hemoptysis, putrid sputum, sputum containing false membrane, etc. It may even be practically pathognomonic, as in the case of "rusty sputum."

*Macroscopic examination* of the sputum is very often sufficient, when taken in conjunction with the other clinical evidences, to indicate the correct diagnosis.

*Microscopic, cytologic examination* yields useful information, but is seldom indispensable.

*Microscopic, bacteriologic examination*, including particularly a search for the tubercle bacillus, is, on the other hand, ordinarily a very necessary adjunct to the clinical examination of the patient. Presence of the tubercle bacillus in the sputum is an indication of an already advanced tuberculous infection of the lung, and all the physician's endeavors and procedures (stethoscopic, fluoroscopic, hematic, etc.) should aim toward the making of a much earlier diagnosis. None the less, the sputum should be examined for tubercle bacilli almost routinely, even in many instances of ordinary acute bronchitis, and in all cases of chronic bronchitis

The tables hereinafter presented will show in a condensed form the various possible types of expectoration, their macroscopic and microscopic features, and their semeiologic value.

Mention should be made of a very simple chemical method of examining the sputum (the albumin test), the exact semeiologic significance of which has not as yet been definitely ascertained (see page 196).

Lastly, reference must be made to a peculiar affection known as Castellani's hemorrhagic bronchospirochetosis, apparently carried into France by Asiatic laborers and soldiers, and studied anew

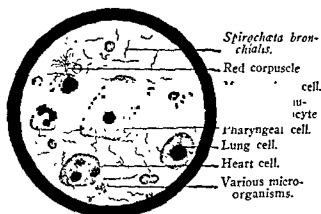



Fig 626.—Smear of sputum in "hemorrhagic bronchitis" Immers object. 1/15 Stiassnie, ocul. 1. Fresh specimen (*Violle*).

during the war by Violle, Dalimier and others (*Presse médicale*, July 5, 1917; July 18, 1918, and Mar. 10, 1919). The condition

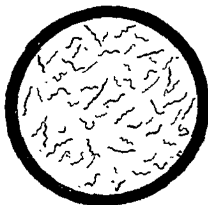


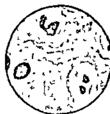
Fig. 627.—Various forms of the *Spirochæta bronchialis*. Immers. object. 1/15 Stiassnie, ocul. 1. Stained by the Fontana-Tribondeau method (*Violle*).

generally consists of a relatively mild bronchitis but one which is accompanied by bloody sputum containing innumerable spirochetal organisms. The period of incubation is short (one to two days);

## SEMEIOLOGY OF THE SPUTUM.

| Macroscopic Appearance | Microscopic Features | | Clinical Significance | Associated Clinical Signs |
|---|---|---|---|---|
| **SEROUS.—Appearance** Frothy, colorless, mucilaginous | | | | |
| | Fibrin<br>Eosinophile cells<br>Alveolar epithelium<br>Sometimes spiral strands (Curschmann's spirals) | | **Chronic serous bronchitis.** | Those of chronic bronchitis |
| **SEROMUCILAGINOUS—Appearance** Like syrup of acacia or whipped cream, sometimes slightly tinted with pink **(albuminoid)** | | | | |
| See illustration above [Serous] | Fibrin<br>Eosinophile cells<br>Alveolar epithelium<br>Sometimes spiral strands | See illustration above [Serous] | (*a*) After too deep or too rapid **thoracentesis.**<br>(*b*) Acute pulmonary edema in chronic nephritis, aortic disease, high pressure cases, heart cases, and pregnant women | (*a*) Those of acute edema:<br>Violent dyspnea<br>Frequent cough<br>Cyanosis<br>Fine râles spreading through the chest<br>(*b*) Those of the causative disease:<br>Albuminuria, high blood-pressure, pregnancy, etc<br>Stethoscopic signs |

**MUCOUS.—Appearance:** Clear, viscid, colorless, often air-laden fluid.—(a) Special variety. Opalescent and highly cohesive, to the point of forming small ovoid or worm-like masses.

| | | | | |
|---|---|---|---|---|
| | 1. Spiral strands (Curschmann's spirals) almost constantly present.<br>2. Charcot-Robin (Charcot-Leyden) crystals. |  | (a) First stage of **acute bronchitis.**<br>(b) At the close of an asthmatic seizure (Laennec).<br>(c) Fibrogummatous **syphilis** (softening). Round or ovoid masses of pea size in the mucoid fluid. | (a) Rhonchi and sibilant râles.<br>(b) Attacks of asthma (see Asthma).<br>(c) History.<br>Sluggish course of the disease.<br>Efficacy of antisyphilitic treatment. |

**MUCOPURULENT.—Appearance:** Yellow or greenish yellow mixture of mucus and pus, or purulent masses in frothy fluid.

| | | | | |
|---|---|---|---|---|
| | 1. Mucin.<br>2. A few epithelial cells.<br>3. A few polymorphonuclears.<br>(Tubercle bacilli in tuberculosis.) |  | (a) **Acute bronchitis** (second stage).<br>(b) **Chronic and subacute bronchitis.**<br>(c) **Acute tuberculosis** (caseous pneumonia).<br>(d) **Chronic pulmonary tuberculosis** (softening and cavities) (nummular sputum). | (a) and (b) Rhonchi and sibilant râles.<br><br>(c) Signs of apical pneumonia.<br>(d) Usual signs of apical consolidation, softening, and cavities. |

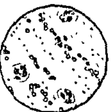
**PURULENT.—Appearance.** Pus, sometimes blood streaked, like pus from an abscess.

| | | | | |
|---|---|---|---|---|
| | 1. Many polymorphonuclears, some showing karyolysis.<br>2. Mucin.<br>3. Streptococci, diplococci, cocci, or rods.<br>(Tubercle bacilli in tuberculosis.) |  | (a) **Cavities** (q.v.)<br>(1) **Pleural:** Empyema (general, interlobar, or encysted).<br>(2) **Pulmonary:** Lung abscess, suppurating hydatid cyst.<br>(3) **Bronchial:** Bronchiectasis.<br>(4) **Subdiaphragmatic abscess:** Hepatic, pyelonephritic, etc.<br>(b) **Pneumonia** in the stage of gray hepatization. | (a) The signs of the causative disorder, plus<br>1. Sharp pain in the side.—Cough.—Dyspnea.<br>2. More or less copious vomiting of pus.<br>3. Immediate relief from symptoms previously present.<br>(b) The usual signs of pneumonia. |

## SEMEIOLOGY OF THE SPUTUM (*continued*)

| Macroscopic Appearance | Microscopic Features | Clinical Significance | Associated Clinical Signs |
|---|---|---|---|
| **PUTRID. FETID. GANGRENOUS.—Appearance:** Fetid odor of gangrene, when set aside separates into 3 layers (a) Upper, mucopurulent —(b) Middle, fluid, flocculent —(c) Lower, greenish-brown | | | |
| | 1 Unrecognizable polymorphonuclears<br>2 Mixture of bacteria | (*a*) 1st degree Temporarily putrid sputum **Temporary fetid bronchitis,** a momentary complication of bronchiectasis.<br>(*b*) 2d degree Constantly putrid sputum **Gangrene of the bronchi.** Curable gangrene of the lungs (bronchiectasis, cavities, etc )<br>(*c*) 3d degree Gangrenous sputum (putrefactive, necrotic, or fecal odor) **Gangrene of the lung.** | (*a*) and (*b*) Physical signs of chronic bronchitis with bronchiectasis<br>Effect of posture on the cough and expectoration<br>(*c*) Physical signs of softening of a lung focus or gangrenous empyema<br>General health markedly impaired |
| **RUSTY, FIBRINO-HEMATIC —Appearance** Muco-fibrinous and viscid, adhering to the receptacle, "rusty" or brick-red, currant or apricot jelly, exceptionally 'prune juice" or "licorice juice" | | | |
| | 1 Polymorphonuclears<br>2 Red cells<br>3 Fibrin and mucin<br>4 Encapsulated pneumococci | (*a*) **Acute lobar pneumonia,** in 9 cases out of 10<br>(*b*) Rare causes·<br>(1) Cancer of the lung<br>(2) Pulmonary infarction (embolism in heart cases)<br>(3) Bloody sputum without infarction in heart cases or in albuminurics<br>(4) Acute pleuropulmonary congestion due to influenza, etc (pseudo-pneumonia) | (*a*) The usual evidences of pneumonia. Pain in side, auscultatory signs, and temperature indications<br>(*b*) The signs of the causative disorder.<br>The sputum seldom presents a definitely rusty appearance, exhibiting features intermediate between rusty sputum and hemoptysis. |

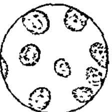
**BLOODY.**—*Appearance*: *Blood distinctly predominates* and is more or less pure, in greater or less amount, and with or without admixture of fibrin or *mucopus*
(*Hemoptysis, q v*)

| | | | | |
|---|---|---|---|---|
|  | Red cells with or without admixture of<br>1 Mucin and *fibrin*.<br>2 Pus corpuscles<br>3 *Various* bacteria (Tubercle bacilli in tuberculosis) |  | (a) Disorders of the *lung*<br>(1) **Pulmonary tuberculosis.**<br>1 *Incipient* — *Initial* symptom<br>2 Established — Recrudescence<br>3 Cavities { Rupture of vessels; Ulcerations<br>(2) Cancer, gangrene, *or* cyst<br>(b) *Disorders* of the circulation<br>(1) Mitral stenosis,<br>(2) Aortic aneurysm<br>(3) Heart failure | (*a*) The usual *stethoscopic signs*<br>Constitutional manifestations Fever, impaired nutrition, *anorexia*, asthenia, sweats, etc<br>Fluoroscopic evidences<br>*Tubercle bacilli* in the sputum<br>(*b*) The usual signs of heart disease |

**PSEUDOMEMBRANOUS.**—**Appearance.** False membrane, a bronchial cast of *natural* size

| | | | | |
|---|---|---|---|---|
| | |  | (*a*) Branching and tubular false membranes<br>(1) Diphtheritic or non-diphtheritic<br>(2) Chronic pseudomembranous bronchitis (rare)<br>(*b*) Whole or fragmented hydatid vesicles (hydatid cyst) | |

**PNEUMONOKONIOSES.**—**Appearance**: Mucopurulent, *this type* of sputum requires separate mention because of its *pigment* content—(*a*) Black *streaks* Anthracosis of coal workers,—(*b*) Red or iron gray Siderosis of workers in iron,—(*c*) Colorless, but sandy Chalicosis of stonecutters

| | | | | |
|---|---|---|---|---|
| | Alveolar cells containing *pigment* or dust particles |  | | |

## SEMEIOLOGY OF THE SPUTUM (*continued*)

| Macroscopic Appearance | Microscopic Features | Clinical Significance | Associated Clinical Signs |
|---|---|---|---|
| **PUTRID. FETID. GANGRENOUS.—Appearance:** Fetid odor of gangrene when set aside separates *into 3 layers* (a) Upper, mucopurulent—(b) Middle, fluid, flocculent—(c) Lower, greenish-brown | | | |
| | 1. Unrecognizable polymorphonuclears<br>2. Mixture of bacteria | (*a*) 1st degree Temporarily putrid sputum **Temporary fetid bronchitis**, a *momentary complication of* bronchiectasis<br>(*b*) 2d degree Constantly putrid sputum **Gangrene of the bronchi**. **Curable** gangrene of the lungs (bronchiectasis, cavities, etc.)<br>(*c*) 3d degree Gangrenous sputum (putrefactive, necrotic, or fecal odor). **Gangrene of the lung.** | (*a*) and (*b*) Physical signs of *chronic* bronchitis with bronchiectasis<br>Effect of posture on the cough and expectoration<br>(*c*) Physical signs of softening of a lung focus or gangrenous empyema<br>General *health markedly* impaired |
| **RUSTY, FIBRINO-HEMATIC.—Appearance** Muco-fibrinous and viscid, adhering to the receptacle, "rusty" or brick-red, currant or apricot jelly, exceptionally "prune juice" or "licorice juice" | | | |
| | 1. Polymorphonuclears<br>2. Red cells<br>3. *Fibrin* and mucin<br>4. Encapsulated pneumococci | (*a*) **Acute lobar pneumonia,** in 9 cases out of 10<br>(*b*) Rare causes<br>(1) Cancer of the lung<br>(2) Pulmonary infarction (embolism in heart cases)<br>(3) Bloody sputum without infarction in heart cases or in albuminuria<br>(4) *Acute pleuropulmonary congestion* due to influenza, etc (pseudo-pneumonia) | (*a*) The usual *evidences of* pneumonia Pain in side, auscultatory signs, and temperature indications<br>(*b*) The signs of the causative disorder.<br>The sputum seldom presents a *definitely* rusty appearance, *exhibiting features* intermediate between rusty sputum and hemoptysis |

Finally, the physician will often be called upon to state "what he thinks" of *some eye condition or other* before the services of an ophthalmologist are sought.

It will therefore not be without utility here to devote a few pages to describing for the practitioner a line of conduct to be followed by him when confronted with a manifest lesion of the eye

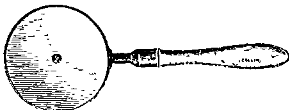


Fig 646—Ophthalmoscopic mirror.

and its adnexa, or with some visual disturbance unattended with any apparent lesion. Furthermore, examination of the eyes is sometimes of assistance in the rendering of a diagnosis and a prognosis as to *vision*, and even as to *life*, in the presence of a general disease.

In any event, after the first visit, or even after a mere conver-



Fig 647—Ophthalmoscopic lens.

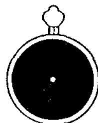


Fig. 648.—Disc with stenopeic opening

sation, the physician must be able to judge IN DUE TIME whether he should seek the help of a specialist. Putting himself in the patient's place, he will have to make up his mind whether he can conscientiously *continue* to treat the patient *alone in a satisfactory manner* "A good physician," said La Bruyère, "is one who has specific remedies, or who, if he has none, allows others who have them to treat his patient." It seems doubtful if any better fundamental

# EYES, DISORDERS OF THE.

## EXAMINATION AND SYMPTOMATOLOGY OF THE EYES.

BY A TERSON, M.D

In his daily practice the non-specialized physician will necessarily be compelled, sooner or later, to see, *first and alone*, diseased eyes and cases of poor vision.

The occasions of this sort vary.[1]



Fig 644.—Lid elevator

Sometimes it is an emergency, medical or surgical, because of *traumatism*, relating to the eye or the rest of the body (with secondary visual disturbance), or because of a rapidly progressive *spontaneous* disorder (suppurative conjunctivitis, corneal ulcer, glaucoma, detachment of the retina, etc ), which compels him to make a *provisional* diagnosis and institute provisional treatment—without postponing too long a special, thorough consultation, at which the necessary accurate conclusions may be reached

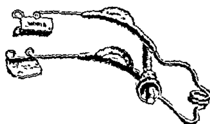


Fig. 645—Terson's eye speculum (blepharostat).

Occasionally some eye complication will develop in the course of another disease which is already under treatment.

[1] For further details the reader is referred to Terson's "*Ophtalmologie du médecin praticien*," 2d Ed., Masson et Cie., 1921.

Obviously, it would be advantageous for the practitioner to measure the *field of vision* and record it on special sheets, and likewise, to probe the *lacrymal passages*, etc. Much experience with these procedures is required, however, if they are to yield conclusive results and be carried out without serious mishaps (injections and catheterization).




Fig 650—Armaignac's test chart, with a special chart for illiterates and the clock-dial chart for the detection of astigmatism

One should have on hand ampoules of 2 per cent and 1 per cent. solutions of cocaine hydrochloride; of 1 · 1000 adrenalin solution; of 1 per cent. pilocarpine nitrate solution, and of 0 5 per cent. atropine sulphate solution Such ampoules, which do not deteriorate and are convenient for carrying about, are frequently just as useful for diagnostic purposes as for the prompt institution of treatment.

rule of professional behavior could be found, even in text-books on medicine.

The practitioner will, of course, undertake only a brief, exoscopic examination, covering mainly that which is visible to the unaided eye. Examination of the eye-grounds, endoscopy of the countless "hidden" disorders of the inner membranes of the eye, and a host of other procedures of mensuration and functional determination are *not* within his province and can be learned only by prolonged training in the living human subject. Among the elementary instruments in the following list are, however, to be found included an ophthalmoscope and its lens,

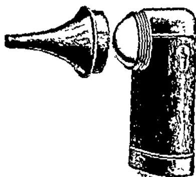


Fig 649.—Electric pocket-lamp with speculum.

the mirror being used *solely* to obtain PUPILLARY REFLECTION, without any attempt to *examine* the *posterior* segment of the eyeball

**Minimum Instrumental Requirements.**—Two lid elevators (Fig. 644); one blepharostat (Fig 645); a simple ophthalmoscope, with lens (Figs 646 and 647); a decimal test chart (Fig. 650) for the purpose of quickly distinguishing normal from defective vision; *three concave lenses of —1, —2, and —3 diopters*, respectively; three convex lenses, of +1, +2, and +3 diopters, and a disc with central stenopeic opening (Fig. 648) (for which a visiting card with a hole put through it may be substituted).

An electric pocket lamp is always serviceable. When combined (Fig 649) with an ear speculum (A Terson), it supplies a sharp beam of light for *pupillary* examination. It may also be used in *diaphanoscopy*.

tient sits, *with his back turned toward the light.* The source of artificial light may be a common lamp, a gas burner (not an incandescent mantle), or a frosted electric bulb. Candle light is generally quite inadequate.

## ELEMENTARY OFFICE EXAMINATION OF THE EYE.

**Preliminary Verbal Examination.**—After having rapidly made note of the general deportment, position of the head (photophobia), and facies of the patient, the physician inquires of him or of his associates (if an infant or child) what caused them to seek medical advice—*e.g*, impairment of vision, ocular or periocular pain, annoying secretions, a structural defect, etc.

Care should be taken to bring out how long the morbid condition of one or of both eyes has been present; what the patient is still capable of in the way of near and distant vision; whether the eyes water and the lids are stuck together in the morning on awakening; whether the pain is constant, intermittent, and spontaneous or *induced* by certain activities or labors; whether the morbid condition began suddenly, rapidly or ran a slow course; whether both eyes began to be affected at the same time, and lastly, whether the disorder is ascribed to some definite CAUSE, *local* or *general* and old or recent

The above verbal examination, *brief* but concise, should be followed by the *local examination*, which is concluded, in turn, by a supplementary verbal inquiry dealing, in addition to any points that may have been overlooked or suggested by the local examination itself, with the present and past medical history and the family history, the latter extending two generations back. The last step consists, where indicated, of a *complete* examination of the patient, including a review of his general health, his habits as regards hygiene, and his mode of life and occupation

**Local Examination.**—The physician first washes his hands thoroughly with soap—in full view of the patient—and then begins with the direct examination, which comprises, as in the case of any other structure, two objective procedures, exoscopy and endoscopy, and thereafter a third, no less indispensable pro-

## I. EXAMINATION OF THE EYE AND ITS ADNEXA.

Even if it is only superficial, an ophthalmologic examination should be carried out *patiently* and *systematically* if any conclusion is to be reached from it. Examination from a point some distance from the eye is insufficient Countless foreign bodies in the conjunctiva and even on the cornea, pupils occluded because of iritis, and cases of chronic glaucoma, granulations, dacryocystitis (even purulent) have been overlooked, *sometimes for months,* because the examiner failed to *get close enough to the eye,* used neither a *lens* nor a *lamp,* and neglected to *evert the eyelids* and to make a direct examination of the eye or the lacrymal sac. And yet innumerable eye washes, useless or actually prejudicial, have been prescribed during these wasted periods.

The observer must *see* the tissues properly with the necessary lens and illumination; he must also adopt means for examining by direct contact the eyeball, the lids, the lacrymal sac, and the region of the orbit, and must check up the functions of *both* eyes, whether the patient is seen at his home or in the office. Mere impressions and suppositions are not sufficient; the physician must be actually certain of anything that conditions permit him to be certain of.

**In the patient's home,** he should have with him the few articles already mentioned for the clinical examination (including a *folding* pocket test chart, mounted on muslin) *Two alternative conditions* may present themselves: Either the patient is unable to get out of bed or he is able to sit on a chair.

If the patient is in bed, the observer should try to have him sit up, and should himself sit on the edge of the bed or on a chair drawn close to it. If the patient cannot be moved at all, *lateral* illumination of the eyes can always be obtained by having some one hold a lamp for the purpose If the patient is sitting in a chair, opposite the physician's, he should be examined first in daylight and then with artificial illumination. The daylight should never stream directly into the patient's eyes, but should reach them *from the side.*

**In the physician's office,** which can be turned into a *dark room* at the appropriate time by means of shades, the test chart should be hung at a distance of 5 meters from the chair on which the pa-

In cases where the fingers do not suffice, lid elevators (Fig 644) or the adjustable blepharostat (Fig 645) should be used. A hair pin bent over at the curved end (Fig. 652) may, *with due care*, be availed of where no other instrument is at hand

In rare instances, instillation of a 1 per cent solution of cocaine hydrochloride (stronger solutions are useless except under certain special conditions) five minutes before the examination is required. The specialist also uses adrenalin under certain definite circumstances The examination of the eye, one region after the other, is then promptly proceeded with

**Eyelids.**—The size and shape of the palpebral fissures of the two sides should be *compared*, the patient opening and closing the

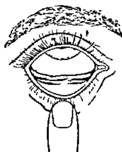


Fig 653—Inspection of the inner surface of the lower lid

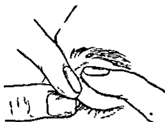


Fig 654—Seizure and eversion of the upper lid (1st step)

eyes Altered direction of the lashes should be looked for with a hand lens

The lids are palpated for nodular masses, which may or may not be tender

The lids should be *everted* in any disorder causing pain. The *lower* lid is merely pulled well down, the patient being meanwhile asked to look *strongly* upward (Fig 653)

Turning the *upper* lid requires more dexterity It must be turned *without causing pain* and *without exerting pressure* on the eye.

**It is absolutely essential that the patient should look down.** He should be instructed to *look at his knees*. The lashes are next seized between the thumb and forefinger of the *left* hand (Fig. 654); the tip of the *forefinger* of the *right* hand then tilts back the tarsus and holds the lid in the everted position (Fig 655).

cedure, *viz.*, examination of the functions of the eye and its adnexa.

The *external examination* (exoscopy) should be made, first in daylight, then by artificial light. For the specialist, the latter method is frequently sufficient.



Fig 651—Examining a child

The older children and adults should be seated in front of the physician; small children and unruly patients in general should be placed with the head low and the extremities held by other persons (Fig 651).



Fig 652—Bent hairpin which may be substituted for the lid elevator in an emergency

In examining the *cornea* and *pupil*—points of major importance in the external examination—no direct contact with the eyelids is required in docile patients and where the eyes give little or no pain In the case of painful and watery eyes, the lids should be carefully dried with absorbent cotton, the observer's index fingers covered with a thin layer of cotton, and these fingers, slightly bent, then applied at the ciliary *margin*. If the fingers are placed *farther in*, the lids come together and prevent inspection of the cornea.

looked, as may, on the other hand, be the case if one merely turns the lid or passes a smooth curet blindly into the cul-de-sac, as text-books invariably recommend.

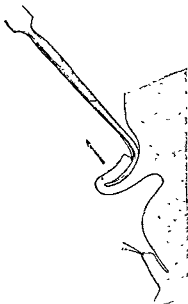


Fig 657—Outward traction of the *already everted* upper lid with an elevator inserted into the fold of skin for the purpose of inspecting the *upper conjunctival cul-de-sac*

**Lacrymal Duct.**—The physician should ascertain whether the lower *lacrymal punctum* is not everted and therefore useless; he should make firm pressure on the lacrymal sac to see if any secre-



Fig 658—Rolling the lid about dressing forceps (1st step).

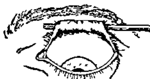


Fig 659—Complete eversion of the upper *cul-de-sac* (2d step)

tion will exude from it (Fig 660) The procedure of making sure of the permeability by *injection* into the lacrymal passages and *catheterization* with an olive-tipped probe belongs to the field of the eye specialist. One may, in any case, instil a *colored* solution such

If the physician's fingers are not small enough for the purpose, the forefinger may be replaced by a probe, a grooved director, a bodkin, or some other thin, smooth object (Fig. 656). If eyelashes are few or wanting, the margin of the lid itself is pinched up.

It is *very necessary* that the patient should CONTINUE TO LOOK STEADILY DOWNWARD during all these manipulations.

When performed gently and methodically, such an instantaneous turning back of the lids causes no pain in a docile patient who *does not insist upon looking upward,* which hinders the procedure.

It is quite useless to attempt to turn the lid with one hand, between two fingers; the procedure already described is preferable and less unpleasant to the patient.

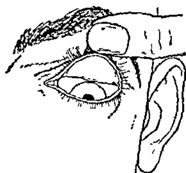


Fig. 655.—Fixation of the everted lid (2d step)

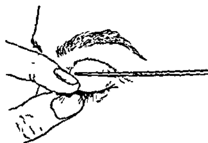


Fig 656.—Eversion of the lid with the aid of a probe.

In some cases it may be necessary to investigate, *in addition,* the upper cul-de-sac, which forms a deep pocket beneath the lid that has already been turned back

Among several procedures available for the purpose, the following are the most effective·

The first consists, after having turned the lid back, in introducing the elevator into the *cutaneous* recess of the lid, which is thus drawn forward, allowing the observer to glance into the cul-de-sac (Fig. 657). The second, or *thorough,* procedure, which EXPOSES the *entire* cul-de-sac, consists, after instillation of cocaine followed by *subcutaneous* injection of the local anesthetic and a *ten minutes' wait,* in having the patient lie down and seizing the lid horizontally with forceps and rolling it about the latter (Fig. 658). With this procedure, no foreign body, tumor, granulations, etc , may be over-

mal shape, etc., and likewise, the *contents of the anterior chamber* (aqueous humor, blood, pus, cellular exudate, etc.), the *outline* of the pupil, the *size* of the pupil, and its *motor responses*, all in the darkened room.

*A comparison of the two eyes should always be made.*

If the least irregularity of the pupil is noted, two drops of 2 per cent cocaine should be instilled; *at the end of half an hour* this will accentuate the peculiarities of shape (from synechiæ, etc.).

**Complete examination of the motor functions of the pupil**—response to light and convergence, or to convergence alone (*Argyll-Robertson pupil*)—has already been mentioned in this work (see

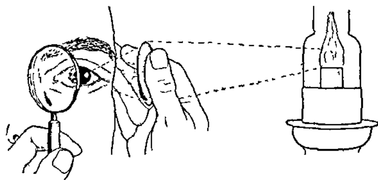


Fig 662—Lateral illumination (examination with two lenses).

p 481), and likewise the manner of eliciting the pupillary reflexes The significance of these tests will be referred to later.

One cannot insist too strongly upon the necessity on the part of the practitioner of informing himself *de visu* as to the actual condition of the *cornea* and *pupil* (adhesion or its absence, and normal or abnormal reflexes) if he wishes to avoid overlooking certain particularly serious disorders (corneal opacity and ulcer, occlusion of the pupil, etc ) which prompt recognition and treatment would have checked before damage hard to undo had resulted

**Palpation.**—Note should always be made of the comparative **tension** of the two eyes The practitioner should confine himself to digital palpation He should *never* press *one* finger against the eye. *Both* forefingers must be used (Fig. 663), and employed in exactly the same way as for eliciting *fluctuation* in an abscess, and after having requested the patient to *look down*. One may then note the

as argyrol or collargol and have the patient blow his nose fifteen minutes later to see if the solution has passed into the nasal cavity, but this gives no information as to the *degree* and *number* of the *stenoses* that may exist

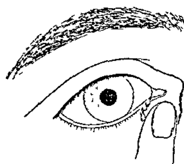


Fig 660—Expressing the lacrymal sac

**Examination of the Eyeball.**—(*a*) CORNEA—The cornea should *always* be examined in a darkened room with *lateral* illumination, concentrated on the eye with a lens (Fig 661); if need be, an additional hand lens may be used (Fig. 662)

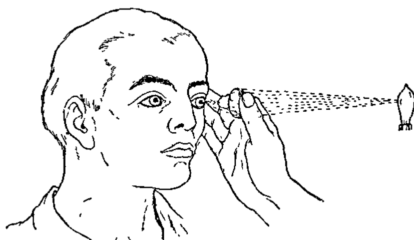


Fig 661—Lateral illumination with a lens

(*b*) CONJUNCTIVA AND SCLERA.—Color, vessels, deformations, etc.

(*c*) IRIS AND PUPIL.—The *surface* of the iris should be carefully examined for fissures, spots, prominence or retraction, abnor-

*lens* (Fig. 665), cellular deposits, *synechiæ* of the iris, and *floating bodies* in the vitreous humor. All these abnormalities appear black, contrasting with the red of the illuminated fundus. At the least movement of the eye, the corpuscles in the vitreous body will be observed *in motion* and thereby distinguished from *fixed* opacities.

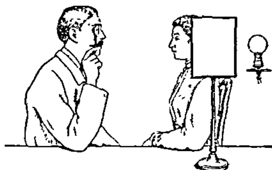


Fig 664—Illumination of the eye with the mirror.

The pupillary field may exhibit several *colors* (partial cataract, dislocation of the lens, swellings, or detachment of the retina).

When a patient states that his vision became impaired *suddenly*, and the pupil appears alternately dark grayish and reddish *under simple illumination with the mirror, without a lens, and with the eye in motion*, one should think of the possibility of detachment of

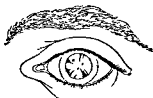


Fig 665—Partial cataract, seen by transmitted light.

the retina, and find out whether the patient sees a hand better in one portion of the visual field than elsewhere.

**Diaphanoscopy.**—The specialist may likewise be led to practice diaphanoscopy, with a lamp (Fig 649) held over the cocainized eye; such an examination is sometimes conclusive (growths, foreign bodies in the lens, etc.).

**Movements of the Eyes.**—With his head *held steadily in one position*, the patient should be required to move each eye in all

firmness or hardness of *increased tension,* which is sometimes stone-like (*glaucoma*), or the *lowered tension* and flaccid condition in certain other disorders.

Pressure may be exerted, if need be, to elicit the *oculocardiac reflex* (see p. 482). In some forms of keratitis, the sensibility of the eye to contact should be tested with horsehair, a fine probe, or a wire previously passed through an open flame.

**Endoscopy.**—The specialist examines the *fundus* of the eye with his perforated mirror and lens. This form of examination,



Fig. 663.—Palpation of the eye for the purpose of estimating the extent of fluctuation

which requires prolonged training and an ability to distinguish the numerous varieties of intraocular disease, is not available to the practitioner who has not gone through the course of studies required for the purpose.

Nonetheless, the practitioner incapable of examining the fundus of the eye may, by illuminating the pupillary region, obtain some information concerning the degree of transparency of the *crystalline lens* and *vitreous body.* The pupil should preferably be first dilated by instilling 2 per cent. cocaine solution, and after waiting half an hour, light is thrown on the eye with the mirror (Fig 664), the patient meanwhile rotating the eye in various directions. There will thus appear, when present, conditions of *partial opacity of the*

turn it in the directions in which he sees the prongs of the letter E pointing on the special test card (Fig. 650).

In some patients vision is so poor that it can be estimated only by the distance at which fingers can be distinguished or a candle seen, etc

(*b*) **Field of Vision.**—Defects in the field of vision show exactly the situation and extent of existing *intraocular lesions;* testing the visual field yields valuable information regarding various conditions

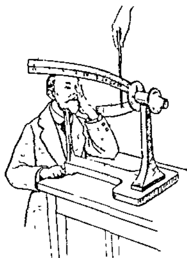


Fig 667—Examination of the field of vision with the perimeter.

of the *brain*, especially through detection of the presence of *hemianopia* The results obtained *localize* the lesions *in the eye without or before their observation by ophthalmoscopy*

Study of the visual field likewise localizes them in the *cranial cavity* and in the *brain*

The field of vision is tested with *the light coming from behind* The campimeter or better the *perimeter* (Fig 667) are the usual instruments employed for the purpose One of the patient's eyes is closed or blindfolded and the examiner moves a white or colored pointer over various points in the field, *at first working in* from the regions inaccessible to the eye The patient says "I see it" as soon as the test object passes into his visual field His head must be held still *throughout the test* and the eye must fix the **central mark**

directions, *the eye not under test being meanwhile covered;* one finds out thus whether there is free ocular motion or motor incapacity. The physician notes whether *diplopia* is present, and if so, whether it disappears (*binocular* diplopia) or persists (*monocular* diplopia) when one eye is covered.

**Orbitoscopy.**—The condition of the walls and accessible contents of the orbit should be summarily ascertained by palpation. If exophthalmus exists, diaphanoscopy, examination of the sinuses, and *fluoroscopy*—the latter also very useful in the detection of intraocular foreign bodies—should be availed of.

**Examination of the Ocular Functions.**—In many instances this may come before any other procedure, even endoscopy.

(*a*) **Visual Acuity.**—Testing visual acuity requires the use of a special test chart (Fig 650), placed at a distance of 5 meters

**T C N D Z P**

Fig 666—A normal eye should be able to recognize these letters at a distance of 5 meters (16⅔ feet)

from the patient, in a good light. If no such chart is available, the practitioner may nevertheless eliminate instances of normal visual acuity if the above line (Fig 666) can be read off with each eye *at a distance of 5 meters, in a good light,* the other eye being meanwhile covered

By means of the test chart, the visual capacity (*acuity*) of *each* eye is ascertained in turn, it is also necessary, however, if vision is *insufficient,* to find out whether this condition is due to a defect of refraction (myopia, hyperopia, or astigmatism) *alone* or *in combination* with some disease of the eye.

Looking through a perforated disc (the stenopeic opening in Fig. 648) or through a pinhole in a card is in itself sufficient to improve vision in persons with defective refraction The remaining procedures (testing with lenses, optometry, skiascopy, etc.) are the particular concern of the eye specialist. The visual power of an illiterate subject or of one with word blindness can be tested by having him hold in his hand a card cut in the shape of a fork and

turn it in the directions in which he sees the prongs of the letter E pointing on the special test card (Fig. 650).

In some patients vision is so poor that it can be estimated only by the distance at which fingers can be distinguished or a candle seen, etc.

(*b*) **Field of Vision.**—Defects in the field of vision show exactly the situation and extent of existing *intraocular lesions*, testing the visual field yields valuable information regarding various conditions

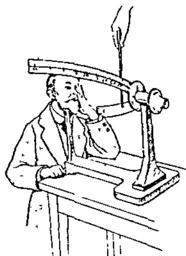


Fig 667.—Examination of the field of vision with the perimeter.

of the *brain*, especially through detection of the presence of *hemianopia* The results obtained *localize* the lesions *in the eye without or before their observation by ophthalmoscopy*

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The field of vision is tested with *the light coming from behind.* The campimeter or better the *perimeter* (Fig. 667) are the usual instruments employed for the purpose. One of the patient's eyes is closed or blindfolded and the examiner moves a white or colored pointer over various points in the field, *at first working in* from the regions inaccessible to the eye The patient says "I see it" as soon as the test object passes into his visual field His head must be held still *throughout the test* and the eye must fix the central mark

directly in front of him. The examiner records on the chart, with a line, the limits obtained in the test and then compares them with the normal field (Fig. 668).

An approximate idea of the visual field may be secured without the special apparatus. The patient keeps his *head motionless, covers one eye* with his hand, and looks steadily at the forehead of the observer, who moves about in front of the eye thus fixed a finger or a piece of white paper held in forceps. He is thus enabled to ascertain whether there exist any *extensive scotomata* (defects)—lateral, superior, or inferior,—or any concentric contraction (retinitis pigmentosa, neuritis, or neurosis) of the field of vision.

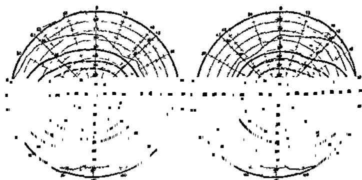


Fig 668—Normal visual fields

The white area represents the field of vision for white The continuous line indicates the limit of the field of vision for blue The line — —indicates the limit of the field of vision for red The line - - - - - - indicates the limit of the field of vision for green.

The **peripheral** defects having first been investigated, there remain to be detected **central** and *paracentral* defects, *positive* **scotomata** (black spots) or *negative* **scotomata** (gaps). The *central* scotoma is the most peculiar type, and is met with in the presence of amblyopia (due to alcohol or tobacco) and disease of the macula lutea.

To make a rough test for central scotoma the patient may be asked to distinguish the color of discs of red or green paper of the size of a ten cent piece or less

Accurate examination of the field of vision is nearly always referred to the eye specialist.

After any examination of the eye and its adnexa, the question of possible *simulation*, a frequent occurrence in industrial accidents, hystero-pithiatism, etc, should, if it arises, be put up to the specialist for solution.

Following is the outline of a *complete* examination of the eye and its adnexa. From among the procedures enumerated the practitioner will select those which he is able satisfactorily to carry out.

**Plan of Examination for an Eye Disorder.**

General appearances: Brief verbal inquiry
Examination (always compare the two eyes)

1. Eyelids, on both their external and internal surfaces The conjunctival culs-de-sac.
2. Lacrymal passages
3. Conjunctiva and sclera
4. Cornea (lateral illumination)
5. Surface of iris and anterior chamber.
6. Pupils: Reflexes and size
7. Crystalline lens, with or without artificial dilatation of the pupil (cocaine)
8. Test of visual acuity (distant vision) and for refractive defects (myopia-hypometropia, hyperopia, and astigmatism).
9. Test of the accommodation and of near vision
10. Test of the field of vision.
11. Test of ocular motility
12. Test of binocular vision
13. Test of color vision
14. Test of intraocular tension and sensitiveness (palpation) The oculocardiac reflex.
15. Endoscopy of the eye (ophthalmoscopy), relating to the lens, vitreous body, choroid and retinal layers, and the optic nerve
16. Illumination by transmitted light (diaphanoscopy) Fluoroscopy and radiography
17. Examination of the orbit (orbitoscopy) and of the periorbital sinuses
18. Special examinations (prospective railway employees, soldiers, or emigrants; industrial accidents, etc) Detection of malingering.
19. Examination of the face and cranium (nose, ears, teeth, lymph-nodes, etc) and of the neck (thyroid gland, etc) A more or less exhaustive examination, as in a patient with some internal disorder (organs in general, heart, liver, urine, blood, comprehensive examination by the latest methods, various tests, blood-pressure, bacterioscopy, biopsy, etc) History. Inherited disorders Supplementary verbal examination
20. Name, age, occupation, address, diagnosis, treatment, date, case number, prescription, and certificate if required

In short, after thorough examination of the eyes a complete examination of the patient is generally necessary in order to confirm the results of the eye examination: After the ophthalmopathy comes the ophthalmopath himself. It is unnecessary here to emphasize how much of added accuracy may be contributed by examination of the cranium, nose, heart and vessels, kidneys, urine, blood and its reactions, etc.—of all the bodily functions and the physical *and mental* circumstances of life—to the *diagnosis*, the *prognosis*, the intelligent and causal direction of *local and general treatment*, and *hygiene* after recovery.

The *local* examination, however well conducted, becomes of conclusive significance only when supplemented by a complete *general* examination, and a satisfactory general examination, in turn, must include an inquiry into all organs and functions, apart from certain exceptional cases.

## II. THE PRINCIPAL OPHTHALMOLOGIC DISORDERS.

Eye disorders may be divided, for the purposes of the unspecialized practitioner, into *two* main groups. In the *first* group, there exists **an obvious morbid condition**, visible to the naked eye, in the ocular and periocular region In the *second*, the patient complains of pain or disturbed vision, but the **eye shows no externally manifest lesion**, or no lesion discernible without some special, complicated *technical procedure*

The following facts will lead to definite recognition or to presumption of certain morbid conditions, while awaiting more enlightened inquiry.

**A Disorders Attended with Externally Obvious Lesions.—Discharges**—(a) *Lacrymation*—Using a *lamp* and *hand lens*, the physician should make a very careful examination of the cornea (erosion, foreign body, slight keratitis, etc ), the iris, the conjunctiva (cul-de-sac and deep aspect of the lids, which should always be everted), the ciliary margin (inturned lashes), and the lacrymal apparatus—in conformity with the directions outlined under *Examination of the eye*—in order to ascertain whether the lacrymation results from some lesion *of the eye* or from a disorder

of the *lacrymal apparatus.* In the newborn, congenital dacryocystitis, nearly always unilateral, is readily mistaken for conjunctivitis if one neglects to compress and empty the lacrymal sac.

(b) *Catarrhal, pseudomembranous, and purulent excretions.*—These result from the presence of catarrhal, pseudomembranous, or purulent conjunctivitis Bacterioscopy is indicated

**Conditions Affecting the Lids.**—Aside from *congenital defects* (coloboma, etc ), there may occur· (a) *Edema·* 1. Passive and *chronic,* in heart disorders, nephritis, etc , or disorders in the vicinity (tumors of the orbit, etc). 2. *Acute:* Urticaria and transient edema

(b) *Infectious edema·* *Local* disorders (furuncles and styes, malignant pustule, erysipelas, subcutaneous abscess, herpes zoster, etc.) or *neighborhood* disorders [lacrymal sac, orbit, sinuses, or eye (conjunctivitis, keratitis, iritis, etc )]

(c) *Emphysema* Comes on abruptly, nearly always upon blowing the nose violently (involvement of the lacrymal passages, nose, and sinuses).

(d) *Ulcerations and tumors,* identical with those affecting the skin in other regions

(e) *Skin disorders* of all kinds, similar to those of the hairy surfaces (BLEPHARITIS ciliaris marginalis) or of various regions of the body

(f) *Abnormal position of tissues*—*Eversion* (ectropion), cicatricial or non-cicatricial *Inversion* (entropion), with or without ingrowing lashes (trichiasis). *Adhesion* to the eyeball (symblepharon). *Tics, spasms, and contractures,* one should carefully ascertain whether the condition is one of purely nervous disease or whether the eye is itself affected (photophobia and blepharospasm in keratoconjunctivitis, folliculo-adenoid conjunctivitis, etc.). *Lagophthalmos* (the eye unable to close in facial paralysis). *Blepharoptosis,* which may be either complete through paralysis of the levator muscle (3d pair), with or without paralysis of the extrinsic muscles; or *incomplete* through paralysis of the cervical sympathetic (Claude Bernard's syndrome, with myosis, enophthalmus, and *slight* ptosis) Ptosis of *conjunctival* origin (*granular* conjunctivitis, etc ) Ptosis merely of *cutaneous* nature, through *dermato-*

*lysis* of the lids One should always examine the pupils, ocular motility, and the condition of the conjunctiva in ptosis.

**Abnormal Conditions and Appearances of the Eyeball**—1. *Congenital anomalies* (anophthalmia, cyclops, microphthalmia, albinism, etc.).

2. *Megalophthalmia* (glaucomatous buphthalmia, extreme myopia, or intraocular tumor).

3. Hypotonic *atrophy* of the globe.

4. Disturbances of *motility* with various types of abnormal positions, with or without oculomotor *paralysis,* and with or without characteristic diplopia (see the table on *Diplopia,* p. 908).

5. *Retraction of the eyeball* (*enophthalmus*)—Due to cachexia and general disorders. *Unilateral* in Claude Bernard's syndrome and certain cases of trauma to the face and orbit. Sometimes *alternates* with exophthalmus.

6. *Prominence of the eye* (*exophthalmus*).—May result from direct *traumatism* (foreign body, hematoma, fracture, etc.) or indirect traumatism (pulsating exophthalmus, sometimes spontaneous); from *inflammation,* as in rheumatic tenonitis (a species of postocular synovitis); orbital abscess; osteoperiostitis in syphilis, tuberculosis, mycoses, or sinusitis; orbitocranial phlebitis or thrombophlebitis (exophthalmus often bilateral); from *tumor, benign* (congenital or hydatid cysts, etc) or *malignant;* or from *exophthalmic goiter* (the *incomplete, unilateral* cases should be watched for).

Examination of a case of exophthalmus necessitates painstaking investigation of *both* eyes and of all their functions, of the adjoining cavities (nose and sinuses, ear, cranium, and mouth), radiography, and a GENERAL examination of the patient.

**Diseases of the Conjunctiva.**—Various *tumors* (*benign,* as in papilloma, pinguicula, etc., or *malignant,* as epithelioma and sarcoma, the latter frequently *melanotic*). Special *dystrophies* such as *pterygium* (fan-like fold of the conjunctiva between the caruncle and cornea). *Lithiasis,* manifested in small yellowish concretions

*Conjunctivitis*—*Secreting*: Pseudomembranous, catarrhal, or purulent. *Vegetating*: Granulations, follicles, etc. *Eruptive*: Pustules, vesicles, and various skin disorders (erythema multiforme, pemphigus, psoriasis, etc.).

One should avoid *confusing* conjunctivitis with *keratitis, scleritis, iritis,* or *glaucoma,* these being much more serious diseases which require prompt treatment of a different kind.

These last disorders are eliminated only by *exclusion.* Close examination can alone reveal, indeed, whether the cornea, pupil, intra-

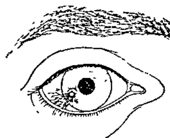


Fig. 669—Pustular (phlyctenular) kerato-conjunctivitis

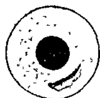


Fig 670—Corneal ulcer

ocular tension, and vision are *normal* Only after having made certain of the integrity *of the other portions* of the eye should the observer look for the distinguishing features of the several forms of conjunctivitis At the same time he should find out whether the

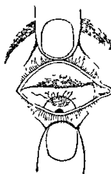


Fig 671—Corneal *pannus* and the upper lid in *granular* conjunctivitis.

conjunctivitis is not *complicated* with keratitis (Fig 669) or some other affection of the eye

The physician should never allow himself to be led into error by faint resemblances to certain disorders

The most important thing in the diagnosis of conjunctivitis is to make sure of the *precise condition of the* OTHER *portions* of the

eye and its adnexa, and actually to see that they are in a normal condition.

**Diseases of the Sclera.**—*General inflammation* (diffuse scleritis) or superficial (episcleritis) or deep (parenchymatous scleritis) *focal* inflammation.

There is less of a bright red color (violet) than in conjunctivitis, and the conjunctiva is smooth, with vegetations or secretions *Congenital* brown *spots* (not to be confused with supra-and intraocular tumors).

**Cornea.**—(a) *Opacity* and *keratitis:* WITHOUT ULCERATION (superficial, parenchymatous, or deep-seated) or WITH ULCERATION (Fig. 670). There should be a detailed examination with the *lamp* and *hand lens.* The observer should ascertain if there is not some

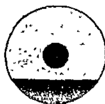


Fig. 672—Hyphema (*blood* in the anterior chamber).

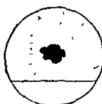


Fig 673—Hypopyon (*pus* in the anterior chamber)

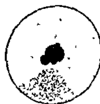


Fig. 674—Iritis with cellular exudation in the anterior chamber.

lesion in the vicinity—dacryocystitis, blepharitis, or conjunctivitis—or a foreign body; if the eye is not *hard* (glaucoma), and whether the opacity is of long standing (cicatricial leucoma) or recent. *Sensation* of the cornea should be tested with a piece of wire, being sometimes abolished (*neuroparalytic* keratitis).

(b) *Vascularization,* a frequent condition during repair of ulcers, congenital syphilitic interstitial keratitis, etc. Vascularization (Fig. 671) of the *upper* portion of the cornea (PANNUS) often points to *granular conjunctivitis* (in such a case the upper lid, seat of the disease, should always be everted)

(c) *Ectasia,* transparent (keratoglobus, keratoconus) or opaque (staphyloma).

(d) *Tumors* about or in the cornea, sometimes melanotic. Confusion with an extravasated intraocular tumor is to be avoided.

**Anterior Chamber.**—Contents *normal* or *abnormal: Hyphema* (Fig. 672), hypopyon (Fig. 673), cell deposits (Fig. 674), foreign

bodies, dislocated lens, etc. The depth of the anterior chamber may be reduced or increased; sometimes it is entirely emptied (the iris being then glued to the cornea).

**Glaucoma** (characterized by intraocular HYPERTENSION).—1 *Acute*: Intense congestion of the eye, paroxsymal pains, pupil *dilated*, eye *very* HARD on *well-conducted* palpation (see *Examination of the eye*), but only slightly sensitive to pressure; vision markedly impaired

It should not be confounded with acute *iritis*. Signs of the latter: Eye red, pupil *contracted* and *irregular*, intraocular tension *normal* or subnormal, palpation very painful, vision interfered with.

2 *Chronic*: But little redness, intraocular tension more or less excessive, field of vision contracted (on the nasal side), pupil *free*

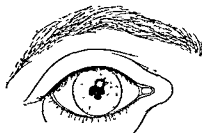


Fig. 675—Syphilitic iritis with granulomatous node

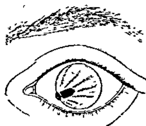


Fig 676—Prolapse of the iris.

*of adhesions* Iridescent vision about lights, such as that of a candle

Instillation of *atropine* is to be avoided where a precise *diagnosis* has *not* been made, for atropine markedly *aggravates* glaucoma—a mistake too often made.

**Ophthalmomalacia**—Transient or permanent hypotonia (with atrophy of the eye).

**Iris.**—*Anomalies*. *Inflammatory conditions* (iritis) and *nodular formations* (granulomata in syphilis, tuberculosis, leprosy, the mycoses, etc.).

The DIAGNOSIS of acute iritis is based: 1. On the *special type of redness* of the eyeball, such redness being especially *pericorneal*, like a *radiating* areola, with *distorted outline* of the narrowed pupil, some parts of the iris becoming adherent to the lens (*synechiæ*);

the pupil appears ragged and FRINGED (Fig 675). 2. On the *subjective* disturbances: *Spontaneous* pain, increased on *palpation*, interference with vision, and photophobia. The upper lid is sometimes swollen. Detailed examination of the *cornea* excludes the existence, or demonstrates the coexistence, of *keratitis*, which causes redness of the same order as that of iritis.

Some cases of *chronic* iritis are not attended with redness nor pain, being featured by the disturbance of vision combined with observation of *pupillary adhesions*

Iritis is distinguished from *acute* GLAUCOMA, first of all, by *palpating* the eyeball, which is HARD in glaucoma; next, by noting that glaucoma exhibits a *dilated*, mydriatic pupil instead of the myotic pupil of iritis. Some cases of chronic iritis may be complicated with increased intraocular tension (*secondary* glaucoma), but examination of the pupil shows the iritic synechiæ.

Iritis should not be mistaken for *conjunctivitis*, in which the redness is *diffuse*, not merely pericorneal, and *the pupil free*, with vision but slightly or not at all interfered with. The secretions, eruptions, and swelling (*chemosis*) complete the diagnosis of conjunctivitis in the absence of any iritic corneal complication.

*Tumors*: Cyst, sarcoma, etc.

*Prolapse* of the iris (in ulcerous or traumatic perforation), forming a small, black, projecting mass.

*Displacements*: Prominence or retraction in certain disorders of the lens and vitreous body. *Tremulousness* (iridonesis) in dislocation of the lens, etc.

**Abnormal Conditions of the Pupils**.—A comparison of the two pupils as to shape, size, and spontaneous or induced mobility should always be made.

One should note the *outline* of the pupil, which is sometimes notched (congenital coloboma), wavy or angular *without* adhesion (glaucoma, tabes, etc.), or irregular and *adherent* to the lens or cornea (*synechiæ*) It is of capital importance to detect these adhesions (*iritis*) of the "fringed" pupil. In doubtful cases, dilatation with cocaine (2 per cent.) will reveal the smallest synechiæ (Fig. 677) after waiting twenty minutes, thus obviating the unpleasant features (visual disturbance persisting for about ten days and interfering with reading) of atropine, which may be reserved for the

treatment of iritis after it has been duly diagnosed. The adhesions of iritis should not be confounded with the threads of a *congenitally persisting* pupillary membrane; the latter do not start from the margin of the pupil, but from the anterior *surface* of the iris

**Mydriasis and Myosis.**—Where uni- or bi-lateral *myosis* is present, one must exclude: The use of a *myotic*, some *disease of the nervous system* (tabes, etc ), *paralysis of the cervical sympathetic* [disease in the *cervical region* (enlarged glands) or in the *mediastinum* should be looked for] with slight ptosis, and *intoxication* by opium, morphine, etc

Where there is *mydriasis*, one must exclude· The use of a mydriatic (sometimes surreptitious), oculomotor paralysis, paralysis of the iris and ciliary muscle (diphtheria, syphilis, etc ), cerebrospinal diseases, lesions of the orbit, and certain intoxications

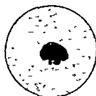


Fig 677 —Synechiæ in iritis

(belladonna, spoiled meat, etc ) The physician should *think of incipient general paralysis* where the pupils are markedly unequal and insensitive to light.

An indispensable proceeding is to ascertain the MOBILITY or IMMOBILITY of the pupil to *light* and *accommodation*, and then to test the *consensual reflex*

The **Argyll-Robertson** pupil (*lack of response* to light coupled with *response* to fixation and convergence) is a sign of nervous syphilis (tabes, general paralysis, etc ), *but very many syphilitics do not show it.*

Finally, there are cases in which.

1 *When light is thrown into* one eye, *its pupil fails to contract, but* the opposite pupil *responds, through the consensual reflex;* the condition then present is a paralytic mydriasis with *peripheral* involvement of the ciliary and pupillary nerves due, *e g*, to syphilis. If light is thrown in the *opposite* eye, the pupil responds, but the pupil of the first eye *remains motionless.*

2. Light is thrown in *a single* eye and *both* pupils remain *motionless.* If now light is thrown in the other eye *alone, both pupils respond.* This condition is the result of a complete blindness of the first eye due to *local* (peripheral) disease of the retina and optic nerve.

In so-called "hysteric" blindness and in blindness of cortical origin the reflexes are retained, as a general rule.

**Pupillary Area.**—The pupillary opening overlies the anterior surface of the lens. The observer should ascertain whether there is not some exudate, with partial or *total* occlusion of the pupil (Fig. 678) and pigment deposits (former iritis).

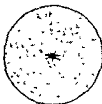


Fig. 678—Complete, *umbilicoid* occlusion of the pupil.

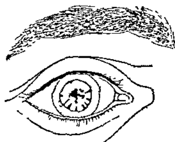


Fig 679—Opacities in the lens (*partial cataract*), contrasting as *black* spots in the pupil illuminated with the ophthalmoscopic mirror

The condition of the lens should be investigated—whether it is *clear* or *opaque* (cataract, partial or total, acquired or congenital, and of many possible varieties).

The observation of a grayish hue of the pupil, very commonly present in old persons, yet unaccompanied by opacity, should not lead to the conclusion that cataract exists The pupil does not seem to be of a clear black when looked at by *daylight;* yet the lens is found to be clear on closer examination.

Aside from the cases in which there is advanced, intense, and unquestionable opacity, as seen *on lateral illumination with a lamp* and by use of the *hand lens,* the physician should not commit himself until *after* he has illuminated the interior of the eye with the ophthalmoscopic mirror, which, after dilatation of the pupil with 2 per cent. cocaine (and waiting 20 minutes), will reveal, contrasting in *black* against the red background of the eye, the slightest lens

opacities (Fig. 679) of incipient cataract—not yet sufficiently marked to prevent reading and writing.

Without being trained in examining the retina and optic nerve, the practitioner may employ for this purpose the mirror and any available source of light, the patient being meanwhile shaded by a screen (Fig 664).

The patient should be requested to move his eye about while the light is being directed at him with the mirror; very often the pupillary area will appear filled with *moving* objects, floating particles, detached retina bobbing about a dislocated and movable lens, crystals, etc

Some eyes that prove UNILLUMINABLE, the pupil continuing to exhibit an ebony black appearance, are the seat of *tumor, hemorrhage, black cataract*, etc

**Luminous Pupil.**—Where, in a child, the pupil presents a glowing, *illuminated* (cat's eye) appearance, there is frequently present a glioma of the retina, a tumor which destroys the eye and is *generally fatal* in spite of enucleation

**B Disorders Unattended with External Lesions, though sometimes Attended with Internal Lesions, Visible only by Endoscopy.** —**Pain.**—Pain in or near the eye, *without any visible pathologic condition*, must be differentiated from headache and migraine—though these are often present in addition.

After having carefully investigated (by *palpation*, etc.) whether the pain is not the result of some *inflammatory condition* which is not yet superficially manifest (beginning stye, keratitis, iritis, overlooked foreign body beneath the lid or in the cornea) or of *increased intraocular tension* (*glaucoma*), the physician should proceed to eliminate simple *migraine* and especially *ophthalmic migraine* with *scintillating scotoma* and images of luminous bands, sometimes with transient hemianopia

He should next think of facial *neuralgia* (making pressure over the points of emergence of the trifacial branches about the orbit) and tic douloureux; a prospective eruption (*herpes zoster*) should be kept in mind. Neurasthenic and hypochondriac patients sometimes experience for years *paroxysms* of ocular neuralgia (delayed *recurrent* keratalgia) in eyes that have previously been subjected

to some form of traumatism, often of trifling degree (erosion from contact with the finger nail or with a house plant, etc.). Very careful examination is necessary in such cases.

PAIN AFTER CLOSE WORK.—*Refractive disorders* (*astigmatism*, hyperopia, presbyopia, and sometimes marked myopia) should be excluded. Headache of this type, which is *very common*, demands the wearing of glasses, in conjunction with general treatment of *coexisting disturbances* (neurosis, anemia, intoxication, etc.). Latent strabismus and insufficiency of and excessive strain in convergence, as well as slight forms of diplopia, may likewise be the cause of the pain

**Miscellaneous Visual Disturbances.—Impaired Vision on One or Both Sides.**—The patient complains of **blurred vision** with **one or both eyes**; or he may be **unable to see anything**, not even light, yet **the eyes seem normal**

Where the patient is still able to see a few objects he should be placed 5 meters away from the test card (Fig. 650), with the latter well illuminated and the patient's back to the light, and his ability to distinguish some of the letters ascertained.

The observer may hold the perforated disc (stenopeic opening) in front of each eye in turn, the *other* eye being meanwhile *covered;* or a card with a pinhole may be substituted If vision is improved thereby, some **error of refraction (myopia, hyperopia, or astigmatism)** is present, with or without disease of the fundus, and the patient will derive more or less benefit from the use of concave, convex, or cylindric *lenses*, which the practitioner may try to select if they are available, but accurate prescription of which requires the intervention of the eye specialist

Where, on the other hand, the stenopeic opening *reduces* vision, **some disease of the fundus** exists, or **an amblyopia** due to a *post-ocular* cerebral or neuropathic disorder.

Here again an examination by the specialist is necessary to settle the question and determine which, among the numerous possible disorders, has been the cause of the blindness or visual impairment in one or both eyes

**Nyctalopia.**—Improvement of vision as the light of day diminishes, occurring in subjects who are *dazzled in broad daylight* (*alco-*

*holic amblyopia*, some cases of cataract, certain diseases of the fundus, etc.).

Hesperanopia.—This term, proposed by the writer, and more accurate than the old term *hemeralopia* (which is etymologically nonsensical), refers to a *sudden reduction* of vision as the light of day declines. The subject *then* becomes nearly blind. There are cases of *transitory* hesperanopia (in overworked or poisoned individuals and disorders of the liver and kidneys) and cases of *permanent* hesperanopia, already present in *childhood* (*retinitis pigmentosa chronica*). It is essential to have the eye-grounds examined in such cases, particularly where the subject is a child who seems helpless and has difficulty in getting about towards evening (*crepuscular* amblyopia).

Phosphenes.—This symptom is an evidence of retinal irritation, which is sometimes of serious degree in *myopic* subjects; it may be a forerunner of detachment of the retina. It is also present in a variety of affections of the retina and choroid. There are even blind subjects who continue to be inconvenienced by luminous visions. The condition should not be confounded with the *paroxysm* of ophthalmic migraine with scintillating scotoma.

Muscæ volitantes.—These are most commonly present in myopia, in overworked individuals, and in neurasthenia. They may sometimes be present for years, or even indefinitely, irrespective of normal vision. Examination of the fundus will show whether the lens, vitreous body, and internal membranes remain in a normal condition and will permit of making a more accurate prognosis.

Disturbances of Color Vision.—These may be *temporary*, as in alcoholic and tobacco amblyopia, affections of the retina and choroid, diabetes, etc., or *congenital*, as in *Daltonism* (color blindness; to be excluded by special tests).

Colored Vision.—Erythropsia (red vision) in neuroses, after cataract extraction, etc. Various phenomena of colored *audition*.

Iridescent Vision.—When a patient sees the colors of the rainbow *when looking at an open flame* (candle), the *intraocular tension* should always be tested, this symptom being often an accompaniment of *glaucoma*.

**Photophobia.**—Where, in the presence of photophobia, the cornea and iris are both found normal, a disorder of the retina or of the lens (cataract) is to be feared. Some neurasthenics, however, are subject to *paroxysms of photophobia* although vision and the eye are and remain normal.

**Vertigo.**—*One should first of all determine whether the patient is not suffering from double vision;* the writer's advice has frequently been sought on account of vertigo attributed to the stomach, previously treated by diet and antidyspeptic remedies, where the actual cause was **paralysis of a nerve or muscle of the eye**

*When a patient complains of vertigo, the physician should* close one *of the patient's* eyes, which will cause *the vertigo to disappear at once if some form of* **ocular paralysis is responsible.**

Vertigo also occurs in connection with deep-seated disorders of the eye, with *unsuitable glasses,* with *overstrong lenses,* and with *general disorders*

**Diplopia and Polyopia.**—One should at once ascertain, by having the patient close one eye, whether he is not *seeing double* **with a single eye;** in some *neuroses,* indeed, and especially in incipient *cataract,* there occurs a **monocular** diplopia or polyopia, the patient seeing, *e g ,* seven or eight gas jets where there is but one, *with one of his eyes*

Where the subject has to *use both eyes* **to see double,** paralysis or contracture of some *muscle* exists, or there may be a mechanical *deviation* of the eye on account of **orbital** disease (tumor or abscess).

A person suffering with diplopia generally shows a *faulty position* of the eye, due to loss of power of one muscle and attraction of the eye by the normally *antagonistic* muscle. *Ordinary strabismus,* or squint, does not cause diplopia, and in spite of the faulty position, each eye, when examined independently, is capable of rotating in all directions; paralysis is not present under these circumstances Faulty position and diplopia are the result of the physiologic peculiarities of the nerves and muscles of the eye (Fig. 680). In *paresis* with but slight muscular weakness, however, or where a single muscle, such as the inferior oblique or inferior rectus, is weak, the faulty position is almost imperceptible, and the localizing diagnosis is based on the diplopia *alone.*

It is well to place a RED GLASS before an eye in order to find out with which eye the red image is seen (*crossed* diplopia—or *homonymous* when on the same side), and to hold a lighted candle 2 meters in front of the patient.

**Objective and Subjective Signs of Paralyses.**—Complete paralysis of the oculomotor nerve (3d pair) which supplies both the superior, inferior, and internal recti, the inferior oblique, and the constrictor of the pupil and levator palpebræ superioris, results in:

*Ptosis, mydriasis, paralysis of accommodation (reading being impossible* except with a strong *convex* lens), divergent *strabismus,* and horizontal crossed diplopia, *i.e.,* a condition in which the *false image* is projected to the *side opposite that of the paralyzed eye.*

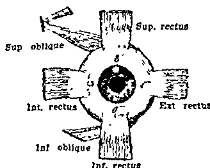



*Fig 680*—*Muscles of the left eye, with their respective distances from the cornea and functions.*

INTERNAL RECTUS, adductor, EXTERNAL RECTUS, abductor, INFERIOR RECTUS, depressor, inward rotator, and adductor, *SUPERIOR RECTUS,* elevator, inward rotator, and adductor, *SUPERIOR OBLIQUE,* with its pulley, depressor, inward rotator, and abductor, *INFERIOR OBLIQUE,* elevator, outward rotator, and abductor

Paralysis of the abducens (6th pair), which supplies the external rectus or abductor muscle, results in convergent *strabismus, with the false image on the same side* (horizontal homonymous diplopia).

Paralysis of the patheticus (4th pair), which supplies the superior oblique or inward rotator, depressor, and abductor muscle, results in vertical homonymous diplopia, with deviation upward and toward the *unaffected* side.

Lastly, there occur instances of independent paralysis of a nerve-branch supplying but *one* of the muscles innervated by the oculomotor, either in ONE EYE or in BOTH, and in which determination of the exact type of involvement is a complex matter. There being no distinct defect of position, the diagnosis is gradually elaborated through *analysis of the existing diplopia.* By moving the candle about before the eyes, at first horizontally and then vertically, *the head meanwhile being kept motionless,* a *progressive* widening of the distance between the images is noted (in the *direction* of the *paralyzed* muscle).

Puech and Fromaget clearly summarize the significance of diplopia in the table reproduced below Examination shows, indeed:

1. *In the* **direction in which the function** *of the affected muscle is normally exerted.*

*False image, increase* of the *diplopia* and *progressive increase of the distance* between the images, *limitation of movement,* an *altered* direction of the *face* and the *inclination* of the *head.*

2. *In the* **direction** OPPOSITE **to the physiologic function** *of the affected muscle*·

Faulty *position* of the eye and *diminution of the diplopia* Later, AFTER having *precisely* determined whether the diplopia is of the HOMONYMOUS OR CROSSED variety, the following

| | | Paralyzed Muscle | Affected Eye |
|---|---|---|---|
| **A.—Homonymous Diplopia.** | | | |
| 1. In the horizontal direction | | External rectus. | The distance between the images steadily increases on the paralyzed side |
| 2. In the vertical direction | above | Inferior oblique. | The upper image corresponds to the affected eye |
| | below | Superior oblique. | The lower image corresponds to the affected eye |
| **B.—Crossed Diplopia.** | | | |
| 1 In the horizontal direction | | Internal rectus | The distance between the images increases in the direction of the motor action of the paralyzed muscle, and hence toward the normal eye. |
| 2 In the vertical direction | above· | Inferior rectus. | The upper image is that of the affected eye. |
| | below | Superior rectus | The lower image is that of the affected eye. |

The general practitioner scarcely needs to undertake such investigations: He may limit himself to the immediate diagnosis of the more easily recognizable forms of paralysis with obvious deviation and muscular weakness—those involving the oculomotor and abducens—and to *blind-folding* one eye to abolish diplopia and vertigo; in *any* kind of paralysis he should then, without further ado, proceed with an investigation of the cause and with causal treatment.

A consultation with the specialist later will determine the further details of the condition, and will also, if the occasion exists, differentiate **paralysis** from **contracture**, which is much less common

**Ophthalmoplegia.**—DIFFUSE forms of **paralysis**, *congenital* or *acquired*, *inherited* and *familial*, of the *nerves and muscles* of the eye.

**Where all the centers (progressive nuclear ophthalmoplegia)** of the motor nerves to the eye fail in succession, the patient, with his drooped lids, with difficulty raised by wrinkling the forehead, and his eyeballs *fixed* in their orbits, is said to present the ophthalmoplegic **Hutchinson's** facies.

Ophthalmoplegic *migraine* (with **sudden and recurrent failure** of all the muscles) is frequently a benign condition, from which *recovery occurs within a few days;* in some instances, however, it is attended with dangerous intracranial disturbances, infectious or neoplastic.

**Paralysis of Associated Movements.**—Cases occur in which, in *both eyes,* whereas other movements of the eyes can still be made, there exists a *paralysis of the associated movements,* **vertical** (paralysis of *elevation* or of *depression* of the eyeball) or **lateral,** owing to disease of the association fibers connecting the centers and convolutions. An ophthalmo-neurologic consultation should be held, as in any other form of paralysis, to determine the cause and seat of the lesion A *complete* examination of the patient should be made, with determination of any co-existing syndromes, such as that of Millard-Gubler (*pontine* lesion with paralysis of the abducens and facial on the same side and hemiplegia on the opposite side) and that of Weber (*peduncular* lesion with oculomotor paral-

ysis and *opposite* paralyses of the face and extremities)—syndromes to be referred to again later in this work.

**Abnormalities of the Visual Field.**—(*a*) PERIPHERAL CONTRACTION.—This may be concentric or excentric and monocular or binocular.

In glaucoma, the patient presents, if the disease is *of long standing*, an *excentric* and INTERNAL contraction of the visual field.

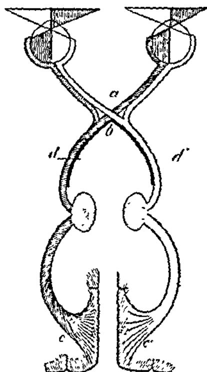



Fig. 681.—Course of the optic nerve-paths (with their *direct* and *crossed* fasciculi) from the eye to the brain, *c*, *c'*, cuneus, *a*, *b*, decussation of the optic tracts (chiasm).

He is unable to see an object placed in front of his eye, and sees it only when it is moved toward the temporal region.

An extreme degree of *concentric* contraction (some fields are only of the size of a dime) may be observed in retinitis pigmentosa, in optic atrophy—*e.g.*, in *tabes*—and in various instances of amblyopia unattended with ophthalmoscopic lesions (hystero-pithiatism, etc.).

(*b*) BILATERAL CONTRACTION WITH OBSCURATION OF ONE-HALF OF THE VISUAL FIELD: **Hemianopia.**[1]—In **hemianopia** there is *complete normalcy of the eye-grounds* and inspection of Fig. 681 will show what the *homonymous* or homolateral (*on the same side*) and the *heteronymous* varieties of hemianopia consist of.

*Central vision is retained* and **the patient is able to read.**

Where hemianopia is suspected on account of the **position** of the patient, *who turns his head* in such a manner as to direct toward external objects that portion of the eye which is still functionally available, and on account of the **absence of lateral vision** of persons, of dishes on the table (the patient *sees his glass, but nothing*

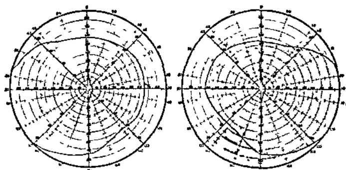


Fig. 682—Right *homonymous* hemianopia the result of disease of the left optic tract at *d* (Fig 681) *or* of its cerebral origin, *c*.

*which adjoins it*), or of the word following that which he is reading, it is easy to **localize** the **intracranial** situation of the lesion responsible This can be done by copying the accompanying diagram and *by interrupting* the optic fibers, either *between the brain and the optic chiasm*, at *d*, for example, showing that there is **homolateral homonymous** hemianopia, right- or left- sided (Fig 682), or in the region of the chiasm, at the *crossed* or *non-crossed* fasciculi, **heteronymous** or **heterolateral**, binasal or bitemporal hemianopia (Fig. 683). There also occur complicated cases, with patch-like defects, atypical forms, double hemianopia, etc.

[1] This condition has often been termed hemio*pia* or hemian*opsia*, the writer prefers to substitute for these the term *hemianopia*, which expresses the *gap* in the field of vision and conforms in its termination to the customary, general nomenclature, as in dipl*opia*, hypermetr*opia*, nyctal*opia*, etc.

For *practical* purposes, *homonymous* hemianopia (Fig. 682) is *nearly* always the result of a cortical lesion in the occipital region and *cuneus* (*softening, syphilitic arteritis, hemorrhage,* etc.). It is generally accompanied by apoplectic strokes, hemiplegia, aphasia, or

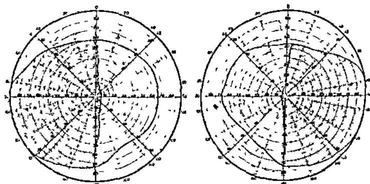


Fig 683—Bi-temporal *heteronymous* hemianopia (in *acromegaly*), due to pressure on the optic chiasm (*crossed fasciculi*)

psychic blindness. Consultation with the *ophthalmologist* and the *neurologist* is quite indispensable.

*Crossed* hemianopia of the *bitemporal* type (Fig 683) is frequently the result of *acromegaly*, the enlarged pituitary body

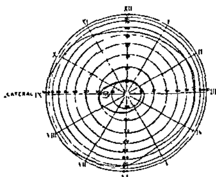


Fig. 684—Central scotoma in an alcoholic subject.

exerting pressure on the *optic chiasm* (*a, b*); whence a characteristic form of visual field (see *Eye conditions met with in general diseases*).

(*c*) Scotomata—The patient complains of *spots* or *gaps* in the central or paracentral portions of the visual field, and not at

its *periphery*. Central scotomata (Fig. 684) indicate defective functioning of the macular region of the retina and are the most annoying to the patient. In some cases they are due to disorders of the fundus, in others to special disturbances, among others *alcoholic* and *tobacco amblyopia.* They impart the sensation of a spot or hole, a *gap* (the patient failing to see certain words in a printed line), or of poor color perception. They should not be confused with *hemianopic defects.*

In all visual disturbances, with or without obvious pathologic change, one should keep in mind the possibility of SIMULATION, which the specialist may be called upon to settle, in common with *artificially induced* disorders of the eye

## III. THE PRINCIPAL EYE CONDITIONS MET WITH IN GENERAL DISEASES.

The majority of diseases, whether acute or chronic, may be attended at some time in their course, sometimes even before their manifest onset, and sometimes during or even after convalescence, by some *organic* or *functional* disturbance of the eye.

It may happen, indeed, that an ocular disturbance appears *a long period ahead,* as the *initial* manifestation of a disorder that is to become obvious and generalized only a long time after Iritis may be the initial localization of a gouty or rheumatic state which will subsequently involve other regions, retinal hemorrhages sometimes precede albuminuria, etc. . The eye condition is thus the *harbinger* of a *latent* general disease In these cases a *complete* examination of the eye patient and inquiry into his family history, together with a *thoroughgoing* plan of *treatment,* calculated to improve all bodily functions and *all conditions that might be considered subnormal,* constitute "the best thing to do" while awaiting the conclusive evidence to be afforded by the subsequent course of events.

Again, while several general diseases are attended by well-known and customary eye complications, these do not always occur; or there may occur others, of very varied type and much more unexpected. To cite one example: Diabetic cataract is familiar to all, but many diabetics do not show it or suffer exclusively from

iritis, amblyopia, and less ordinary eye affections; *yet these are characteristic* to an ophthalmologist.

Finally, assuming a loftier view, one may recall how the assimilation and sometimes the *identification*, in GENERAL AND COMPARATIVE MEDICINE, of the ocular disturbances—*regional* states comparable to those observed in other portions of the body and in other organs—is a matter of compelling interest and affords a solution for many *ophthalmologic* or *general* problems which could not be solved by men restricted to their own special field, medical or surgical. As far as the writer is concerned, he has always believed and taught, through the last twenty-five years, that an eye condition is, as a rule, in all respects, even as regards treatment, an *oculo-general syndrome*. There is no doubt but that the other specialties would derive benefit from a similar, daily *confrontation* of observations, as well as from a like *joint conclusion* as regards the etiology and treatment.

**Diseases of the Kidneys.**—Many cases of *Bright's disease* remain free of ocular disturbances throughout. One should be wary as regards coincident disorders (hyperopia, myopia, etc.), *non-*pathognomonic complications such as iritis, cataract, paralyses of eye muscles, etc., and those due to a concomitant disease, and should have a complete examination of the eyes made in these cases.

*Edema of the lids*, bilateral and of varying degree (less common than is generally believed), *retinitis* with hemorrhages and *white spots*, accompanied by *edema of the optic nerve*, are more characteristic. The patient has difficulty in reading or cannot read at all, but can still get about Blindness is rare, *unless some complication occurs* such as detachment of the retina, glaucoma, etc. *The eye disease is recovered from, however, only if the nephritis is likewise cured.*

Retinitis sometimes precedes the appearance of albuminuria (*prealbuminuric* retinitis) in patients with arteriosclerosis and high blood-pressure.

It occurs frequently in combined *glycosuria and albuminuria.*

Retinitis with white spots is rather the result of the *azotemic* condition; edematous neuro-retinitis, of *chloridemia*, and the retinal hemorrhages, of *high blood-pressure*. These several types, while sometimes present independently, are rather usually *combined*.

Thus, the patient almost never becomes blind, but in the many cases of *incurable* nephritis the appearance of retinitis is an *unfavorable omen.* Statistical records show that "ocular" nephritics, with few exceptions, die sooner, generally *not surviving longer than two years.*

Some patients with albuminuria and high blood-pressure suffer *attacks of transitory amaurosis,* unaccompanied by disease of the eye-grounds and followed by return of vision.

**Diabetes.**—Aside from retinal hemorrhage and mixed forms of retinitis (diabetes with albuminuria), diabetic patients may suffer from cataract of different varieties (soft, white, and bilateral in young subjects) and more or less operable under local and general precautions; acute or chronic iritis, hemorrhagic glaucoma; amblyopia with central scotoma (of the toxic type, similar to that in alcoholic amblyopia), optic atrophy; paralysis of the motor nerves of the eye, rapidly progressive myopia, etc. The urine should always be examined in eye disease, especially when deep-seated

**Gout.**—Iritis, sometimes hemorrhagic; scleritis or sclerotenonitis

**Arthritis Deformans.**—Iritis, iridochoroiditis, scleritis, or anterior sclerochoroiditis

**Diseases of the Heart and Vessels.**—(*a*) Heart Disorders.—In the last stage, edema of the lids, the optic nerve, and the orbit; sometimes retinal hemorrhages, glaucoma, and hemorrhagic glaucoma Embolism and thrombosis of the retina in chronic endocarditis, causing *sudden blindness on one side,* which is often permanent.

Pulsation of the *retinal vessels* is frequently present in aortic insufficiency and at times in mitral insufficiency.

Spontaneous subconjunctival ecchymoses, sometimes preceding cerebral hemorrhage, occur in arteriosclerosis and high blood-pressure.

Inequality of the pupils is observed in aortic aneurysm (pressure on the sympathetic)

(*b*) Major hemorrhages.—Certain profuse hemorrhages, as in *hematemesis,* intestinal hemorrhage, *metrorrhagia,* epistaxis, wounds, etc, lead either at once or later, *several days after* the loss

*of blood,* to bilateral disturbances of vision which may go on to *blindness,* partial or complete, curable or incurable (complete or partial optic atrophy).

(*c*) BLOOD DISTURBANCES.—A *thorough* examination of the blood *by the latest methods* is of exceedingly great importance in the majority of diseases of the eyes and their adnexa, both as regards diagnosis, prognosis, and treatment. One should not be too positive in concluding that syphilis is absent where the Bordet-Wassermann reaction is *negative;* in the writer's experience, intensive antisyphilitic treatment has often brought about a recovery under these circumstances in a wide variety of eye disorders. An investigation for syphilis should always be made, even in the presence of manifest tuberculous disease, which is often combined with syphilis.

A study of the blood should be made in chlorotic anemia, leukemia, Hodgkin's disease, hemophilia, etc, where localized ocular involvement exists

**Various Infectious Diseases.—Eruptive fevers.**—MEASLES.—Conjunctival hyperemia, chiefly in type (like pterygium); styes, abscess of the lid, pustular keratoconjunctivitis, blepharitis or dacryocystitis.

SMALL-POX AND CHICKEN-POX.—Pustular formations in or about the eye, serious *corneal ulcers* and *abscesses;* iritis, iridochoroiditis, or optic neuritis, orbital abscess; chronic ulcerative blepharitis with misplaced lashes Persons in whom a *vaccinal* eruption is at its height should be cautioned not to touch their eyes with their fingers (vaccine blepharitis and keratitis)

SCARLET FEVER.—Conjunctivitis, sometimes with false membranes; dacryocystitis, albuminuric retinitis.

*Note.*—In all eruptive fevers with eye or lid involvement the physician should ascertain *daily,* with the LAMP and HAND LENS, the *exact* condition of the *cornea* and *pupillary margin.*

DIPHTHERIA.—Paralysis of accommodation with mydriasis; the patient is unable to read, unless a strong convex lens of +3 or +4 diopters is used, or a 1 per cent. solution of pilocarpine nitrate is instilled and he waits half an hour for its effect. Paralysis of eye muscles; pseudomembranous conjunctivitis

The *pupils* and *vision* should always be examined in the presence of any kind of *sore throat.*

GRIP.—Herpes of the cornea, transient or lasting; iritis, etc Grip initiates, relights, or aggravates the majority of eye disorders.

MUMPS.—Inflammation of the lacrymal *glands* (dacryoadenitis); conjunctivitis, iritis, or optic neuritis.

TYPHOID FEVER —Optic neuritis, paralysis of ocular muscles, etc.

MALARIA.—Obstinate herpes corneæ, iritis, retinitis, floating particles in the vitreous humor, optic neuritis.

ACUTE AND RECURRENT RHEUMATISM.—Iritis, scleritis, or sclerotenonitis; causal rôle in other eye affections more doubtful

ERYSIPELAS —Abscess of the lid, dacryocystitis, conjunctivitis, corneal ulcer, iritis, optic neuritis; phlebitic orbital infections. Sometimes a ("curative") erysipelas causes *improvement* in a rebellious eye affection (granular conjunctivitis, leprosy, etc.)

ANTHRAX —Malignant pustule and edema of the lids

LEPROSY —Leprous nodules of the lids, conjunctiva, or cornea; iritis; paralysis of the orbicularis muscle

TUBERCULOSIS —Almost any of the syndromes—whether *inflammatory* or in nodular *foci*—affecting the eye and its adnexa may be of tuberculous origin. One should exclude or detect syphilis, acquired or congenital, and the other affections frequently present in combination

SYPHILIS —Chancre of the lids or conjunctiva Pustules, papules or patches• Gummas and syphilomas of the lids, conjunctiva, lacrymal sac, lacrymal gland, orbit, cornea, sclera, or *interior of the eye* (*perforating* gumma of the iris and ciliary body) Intraocular inflammatory states, acute or chronic (iritis, choroiditis, retinitis, optic neuritis, etc.) Optic atrophy, with or without tabes. Paralysis of oculomotor muscles. Frequent anachronisms in the various manifestations (early tertiary or delayed secondary manifestations).

Syphilis in its *acquired* or *congenital* forms should always be looked for in the presence of ocular disorders *Any* of the aforementioned eye conditions may be met with in congenital syphilis, with a special predilection, however, for *interstitial* keratitis, chorioretinitis, congenital cataract, buphthalmus (infantile glaucoma), strabismus, and nystagmus. Tuberculosis is frequently present in association The *therapeutic test* by means of antisyphilitic treatment—which should not be neglected in spite of the results of the

blood or other tests, whether positive or negative—oftentimes yields unexpected benefit.

MYCOSES.—A definite differentiation of these disorders from syphilis, tuberculosis, and the other infections of the eye and its adnexa should be made

**Diseases of the Respiratory Tract.**—(*a*) NOSE.—*Dacryocystitis*, lacrymal stenosis in the presence of rhinitis and *ozena*, with or without serious *corneal ulcers*. Adenoid growths are often present in combination with follicular conjunctivitis (likewise an *adenoid* condition) and with pustuloscrofulous (or *phlyctenular* or *impetiginous*) conjunctivitis. Exophthalmus may be produced by extension of nasal tumors.

(*b*) SINUSES.—Many ocular and periocular conditions develop in *sinusitis* (optic neuritis, iritis, detachment of the retina, orbital abscess and orbitocranial phlebitis, paralysis of ocular muscles, etc.).

Examination of the nose and sinuses is imperative in most affections of the eyes and especially in affections of the lacrymal passages and orbit, just as examination of the eyes is necessary in disorders of the nasopharynx and sinuses.

(*c*) LARYNX, TRACHEA, BRONCHI, AND LUNGS.—*Pupillary* disturbances (mydriasis or myosis) may occur in cases of tumor or enlarged glands through paralysis or irritation of the sympathetic (slight ptosis, enophthalmus, and myosis in *Claude Bernard's syndrome*). *Mydriasis on the side of a diseased lung (pleurisy*, pleural pneumonia, tuberculosis, etc.) is rather common. Consumption generally runs its course, however, without any special eye disturbances.

Herpes corneæ, iritis, and optic neuritis occur in influenza and pneumonia.

An *extensive* subconjunctival ecchymosis sometimes occurs in whooping-cough, but is *unattended with risk*, no hemorrhage in the fundus of the eye taking place.

**Diseases of the Ears.**—Iritis, choroiditis, optic neuritis, orbitocranial phlebitis, paralyses of ocular muscles—especially the abducens distribution—are met with in otitis and mastoiditis; nystagmus, spontaneous or induced, may be encountered.

**Diseases of the Digestive Tract.**—(*a*) TEETH AND MOUTH.—*Infections*, such as orbital abscess and phlebitis, iridochoroiditis,

optic neuritis and keratitis *Mixed* or *reflex* complications (neuralgia, amaurosis, blepharospasm, glaucoma, paralyses of ocular muscles, etc.). *Coexisting disorders* may be exemplified in the *oculodental* stigmata of congenital syphilis, rickets, etc. Mikulicz's syndrome, involving the lacrymal and salivary glands.

(*b*) ESOPHAGUS.—Tumors, resulting in Claude Bernard's syndrome.

(*c*) STOMACH AND INTESTINE.—Enterogenous autointoxication and intestinal infection take part in very many eye disorders. Blindness, transient or permanent (optic atrophy), may follow hematemesis or intestinal hemorrhage. *Intraocular* metastases of visceral cancers. Ocular parasites of intestinal origin.

(*d*) LIVER.—Choroiditis, chorioretinitis, retinal cirrhosis (retinitis pigmentosa with *hesperanopia*), or retinal hemorrhages. Xanthoma of the lids, conjunctival icterus, or lithiasis conjunctivæ.

Metastases in the liver are frequent in melanosarcoma of the eye

**Diseases of the Skin.**—ANY *skin disorder* may occur primarily or secondarily on the *eyelids;* many occur likewise on the *conjunctiva* and even on the *cornea* Similarly, diseases of the *hair* may occur in the *eyebrows* and *eye-lashes.*

*Blepharitis ciliaris* or marginalis corresponds to the various types of skin disorder, *viz*, eczema, seborrhea, folliculitis, sycosis, etc, the exact nature of which should be inquired into in each individual case

Among the most noteworthy general pathologic conditions, special mention should be made of *erythema multiforme,* with its huge conjunctival papules directly on the *eyeball,* of *acute cutaneous edema,* sometimes alternating with acute *glaucoma*—further proving that acute glaucoma is a condition of *acute edema* occurring in the eye and associated with increased intraocular pressure (A. Terson), and of *pemphigus,* which gradually results in complete fusion of the eyelids with the immobilized ocular globe (symblepharon).

**Diseases of the Ductless Glands.**—(*a*) *Thyroid gland.*—Exophthalmic goiter, complete or fruste (incomplete or truncated; unilateral, dissociated, etc.), with exophthalmus, Graefe's sign (delayed motion of the upper lid when the patient looks down), tachycardia and tremor. Sometimes there are inequality of the pupils,

paralyses of ocular muscles, etc. Reduction of pulse-rate on testing the oculocardiac reflex.

The condition of the *cornea should be watched* in marked *exophthalmus* (the cornea being examined with a *lamp* and *hand lens*) in order to detect any *incipient* ulceration and to be able to apply the *emergency* treatment (tarsorrhaphy, etc.).

(*b*) *Adrenal glands.*—Asthenia of the eyes, etc.

(*c*) *Pituitary body.*—Exophthalmus, nystagmus, and paralyses of ocular muscles in conjunction with the acromegalic facies. Progressive optic atrophy.

The fields of vision should always be tested—*bitemporal hemianopia* with loss of the two *outer* halves of the visual fields on account of pressure on the two crossed fasciculi of the optic nerve at the optic chiasm (see Fig 683)

(*d*) *Testicles and ovaries.*—Effects on the eyes may be of the asthenic, hypersthenic, or toxic types,—to be determined with a varying degree of probability in the individual case.

**Diseases of the Reproductive System.**—(*a*) MALE—Gonorrheal ophthalmia, direct or metastatic, with scleritis, iritis, optic neuritis, or dacryoadenitis.

(*b*) FEMALE.—Iridochoroiditis and deep-seated infections of metritic origin, following the menopause, etc. Menstrual asthenopia. Optic atrophy following profuse metrorrhagia

Retinal hemorrhages and neuroretinitis during pregnancy, the nephritis of pregnancy (sometimes necessitating induction of labor), or lactation. Pulsating exophthalmus. Metastatic intraocular suppuration (puerperal sepsis).

Purulent conjunctivitis of the *newborn.* Serious accidents to the eyes (exophthalmus or orbital fracture) due to use of the forceps or various other obstetrical procedures.

**Diseases of the Nervous System.**—HYSTEROPITHIATISM.—Amblyopia and amaurosis, with sudden blindness, total or partial. The eye-grounds are normal and the pupillary reflexes are preserved—Concentric contraction of the visual field with inversion of the color fields. In some instances, anesthesia of the conjunctiva, blepharospasm, facial hemispasm, strabismus, or spasm of accommodation (special, transient myopia).

Examination of the eyes is imperative, to obviate confusion (organic disturbance or simulation).

NEURASTHENIA.—Ocular asthenia (asthenopia) while at work; muscæ volitantes, ophthalmic migraine, or periocular neuralgia; transitory contraction of the visual field due to fatigue.

EPILEPSY.—Frequency of refractive defects and eye disorders, stigmata of inherited syphilis, alcohol and tobacco amblyopia, etc.

INSANITY.—Attacks of ocular hyperemia. Auto-mutilation of the eye, in several instances extending to the rapid tearing out of *both* eyes, the preliminary incision about them being made with the sharp finger nails.

IDIOCY.—Stigmata of inherited syphilis; congenital anomalies.

MENINGITIS.—Mydriasis or myosis. Optic neuritis. Iridochorioretinitis. Tubercles of the choroid. Paralyses of ocular muscles. Total or partial optic atrophy. Nystagmus. Strabismus.

INTRACRANIAL THROMBOPHLEBITIS.—Exophthalmus, generally bilateral. Paralyses of ocular muscles. Optic neuritis.

ENCEPHALITIS OF VARIOUS TYPES.—Lethargic encephalitis, among others, exhibits paralyses of the ocular muscles in successive groups or "waves;" paralysis of associated movements (convergence); ptosis; nystagmus.

HYDROCEPHALUS.—Optic neuritis and atrophy; paralyses of ocular muscles; nystagmus and strabismus. Coexisting signs of congenital syphilis or tuberculosis.

BRAIN SOFTENING, CEREBRAL HEMORRHAGE, ETC.—Amblyopia; word blindness; hemianopia, *usually homonymous*, permitting of localization of the disease in the hemisphere *opposite* to that of the field defects (see Fig. 682).

TUMORS.—Edema and venous congestion of the optic nerve owing to increased intracranial pressure. Paralyses of ocular muscles. Hemianopia. These signs, while frequently present, may be lacking or present with conditions other than tumor. They confirm the diagnosis of endocranial tumor, but *seldom suffice* in themselves to *localize* it.

INVOLVEMENT OF THE CEREBELLUM, PEDUNCLES, OR PONS.—Syndromes of Millard-Gubler or of Weber (p. 909), in conjunction with other localizing neurologic syndromes; optic neuritis.

MYELITIS.—Optic neuritis (optic neuromyelitis), sometimes preceding the myelitis.

TABES DORSALIS.—Argyll-Robertson pupil; pupillary inequality; frequently myosis Irregularity of the pupillary *outline* (*oblique-ovaloid* pupil of A. Terson). Sclerous atrophy of the optic nerve, as yet incurable and becoming bilateral in spite of the latest forms of treatment. HIGHLY *intensive* treatment further *accelerates* the reduction of vision, notwithstanding the fact that these patients are known to be syphilitic. Paralyses of ocular muscles, transient or persistent.

GENERAL PARALYSIS.—Inequality and irregularity of the pupils with *mydriasis* and paralysis of the iris. Toward the final stage, optic neuritis and atrophy, and motor paralyses.

FRIEDREICH'S ATAXIA.—Nystagmus; sometimes optic atrophy.

LITTLE'S DISEASE.—Strabismus; nystagmus; stigmata of inherited syphilis.

DISSEMINATED SCLEROSIS —Nystagmus; paralyses of ocular muscles; rarely, sclerosis of the optic nerve.

**Intoxications.**—ALCOHOL AND TOBACCO.—Amblyopia, bilateral from the start, with central scotoma.

*Far* vision is impaired, but the subject, while *dazzled* by bright light and nearly blind in broad daylight, *has much better vision in the evening* (crepuscular improvement).

*Near* vision is greatly interfered with; the patient is unable to read, and distinguishes certain colors poorly (*green* and *red*), especially over a small area, on account of the central scotoma. While he recognizes the color of a *sheet* of red paper, he is unable to state the color of *disc* of red paper of the size of a dime. When the disease is sufficiently advanced, he *mistakes a dime for a silver dollar.*

This condition, if taken at the start and treated by a strict diet and suitable remedies, may be *completely* recovered from if the subject does not resume his harmful habits.

The practitioner should always have an examination of the eyes made in order to exclude certain VERY *serious mistakes* (diabetic amblyopia, macular chorioretinitis, retrobulbar neuritis, optic atrophy, etc.) and diagnose coexisting disorders.

Many other forms of *poisoning* (lead, male fern, pelletierine, iodoform, etc.) may give rise to toxic neuritis and a variety of ocular and oculomotor disorders of a lasting or transient nature.

Ingestion of large amounts of *quinine* at a single dose sometimes induces temporary blindness, which terminates either in recovery or in partial and lasting sclerosis of the optic nerves.

*Atoxyl* has been the cause of many cases of optic atrophy and incurable blindness.

*Naphthalene*, when ingested, yields an experimental form of cataract (in animals).

*Botulism* may bring about optic neuritis and paralyses of the ocular muscles, including those of accommodation.

**Ophthalmic Signs and Reagents Indicating Death.**—Examination of the eye is of assistance in distinguishing *actual* from *apparent* death and in obviating premature burial, whether under ordinary or unusual circumstances (wars, epidemics, or catastrophes).

Loss of the winking reflex, loss of corneal sensibility, dilatation of the pupil following myosis (frequent in the agonal state), a dull appearance of the eyes, and complete opening or closure of the lids (a very variable condition in different subjects and according to the manner of death) constitute merely presumptive evidence. The *pupils* of a *corpse* will *often* react *for several hours*, especially to myotics (eserine and pilocarpine) and electric stimulation Palpation of the eyes may be practised; they become particularly flaccid at the end of several hours.

In S Icard's procedure (subcutaneous staining injection of fluorescin, which fails to diffuse in a *dead body*), the eye may assume a greenish tint in a *living* subject; but this phenomenon is very inconstant.

Lecha Marzo has made an investigation such as had already been made in other regions of the body, of the existence of a *post-mortem acid reaction* of the tears, which are neutral or alkaline in the living subject, by placing a piece of litmus paper beneath the eyelid. Unfortunately the time of appearance of the acidity, as well as its intensity, are highly variable after *actual* death. Distinct acidity, it is claimed, is never present, however, in a living subject in a condition of *apparent* death

*Instillations of irritants* and *mechanical* stimuli (scraping or chemical or actual cauterization of the conjunctiva or of the *upturned* lids, etc ) may be tried, and will induce redness and hyperemia in an inert *living* subject. Instillation of ether (d'Halluin) may, however, not be without risk to the cornea. The writer prefers the introduction of a wheat-grain-sized amount of powdered ethyl-morphine hydrochloride (dionin), a substance used in daily practice without harmful results, and which induces redness and swelling, often of considerable extent, of the conjunctiva in the eye of a living subject.

One need *not limit himself* to the ocular tests of death, but they should be utilized among the *routine* measures for the determination of death.

The foregoing summary considerations on the subject of combined ophthalmology and general medicine may have served at least to suggest to the practitioner the marked importance of an objective and functional examination, whether positive or negative, of the eyes and their adnexa in the diagnosis and prognosis of almost any disorder affecting the human body—not to mention the cases in which such an examination, as yet so frequently neglected or inadequately performed, actually permits of apprehending, arresting, or curing a disease of the eye itself.

## FAINTING (SYNCOPE).

[*From σύν, with, and χόπτειν, to cut. Faintness, fainting spell.*]

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True syncope consists of a sudden, temporary cessation of the heart's action. It is exceedingly uncommon. On the other hand, faintness, semifainting, or lipothymia (from λείπειν, to relinquish, θυμός, spirit) is met with rather often, and is characterized by a more or less complete loss of consciousness apparently dependent upon a reduction of varying degree in the blood flow (ischemia) through the brain. The nervous and circulatory systems are so intimately interdependent that psychic and circulatory manifestations are in close association, and the most reliable sign of syncope and of lipothymia, which sign. moreover, allows of their immediate differentiation from coma, asphyxia, etc., is the combined observation of a more or less complete loss of consciousness with weakened and sometimes slowed heart action, the latter sometimes passing into actual cessation of the heart beats for a varying period of time. Syncopal states, then, are characterized

1. By a more or less profound state of fainting and unconsciousness, with more or less complete muscular relaxation.

2 By a marked weakening of the pulse (small, feeble pulse) and of the heart beats.

3. By certain associated vasomotor and secretory disturbances, *viz*, pallor of the face and lips, cold sweat, cold extremities, etc, which impart in some degree to syncope the appearances of death.

It should be noted that frequently, just before the termination of syncope, a short general convulsive seizure is observed, independently of any epileptic tendency.

Thorough realization of the three above mentioned characteristic features will suffice to eliminate, usually at the first glance, artificial fainting spells, "theatrical faints," and "suggestive syn-

copal states" so prevalent in certain quarters, and seemingly in all historical periods, judging from the high percentage of "swoons" referred to in the romantic literature of all ages and all climes. "Clarisse Manson," wrote Lenôtre (the celebrated witness of the Fualdès episode), "succeeded in holding in anxious suspense the attention of the entire world for two whole years merely by swooning, a proceeding which she carried out to perfection and repeated indefinitely without becoming fatigued."

Syncope and syncopal states are met with chiefly under the following conditions:

1. **Ordinary fainting,** probably due to acute anemia of the brain (by vaso-constriction or nervous cardiovascular inhibition) acting upon *predisposed individuals* when in a confined or poorly ventilated room, in the presence of a crowd, or unexpectedly witnessing some accident or a hemorrhage. The sight of blood-shed regularly causes faintness in some persons who may properly be said to be *hemophobic,* and in these individuals personally experienced pain or hemorrhage has, of course, an even more certain effect in this direction. The wearing of a corset or other tight garment, and either the period of digestion or that of fast, plainly predispose to fainting in some persons. In short, at the bottom of the condition there are always found: 1. Some emotional impression (anxiety, apprehension, pain, fear, terror, etc.) 2. An emotional neurocardiovascular predisposition, finding its ultimate expression in an exaggeration of the nervous vasomotor and inhibitory reflexes.

Allied to ordinary fainting are the faintness and lipothymic attacks of patients with low blood-pressure, of convalescents, of septic cases, and of cases of visceroptosis, accounted for in each case by a manifest state of neurovascular weakening.

2. **Certain minor forms of epilepsy** are commonly considered related to ordinary fainting, but in these cases the vasomotor manifestations characteristic of syncopal states, *viz.,* small pulse, coolness of the extremities, etc., are, as a rule, absent. Yet it must be admitted that some of these conditions are very similar to syncope. They should always be thought of in the presence of recurring pseudolipothymic attacks or repeated faints of obscure causation.

3. Certain cerebrocardiac forms of arteriosclerosis, especially if accompanied, as is the rule, by aortitis, may lead to particularly dangerous syncopal states. The fatal syncope of the major forms of angina pectoris belongs in this group. It is mainly the possibility of such an occurrence that furthers an unfavorable prognosis in the latter disorder Yet it should be mentioned and repeated that even in the major forms of angina pectoris, even in the presence of definite and extensive aortic lesions, such as dilatation or actual aneurysm, and even with a rather marked elevation of blood-pressure, fatal syncope is exceptional and, in any case, perhaps, is generally long delayed. The author has had, and still has, under observation such subjects for periods of ten or twelve years or over.

For practical purposes the following general rule may be adopted:

*Syncope is ordinarily a mild condition in young individuals; on the other hand, it is always a serious, at times a dangerous, and sometimes even a fatal, occurrence in old persons*

4 The fainting of paroxysmal bradycardia (Stokes-Adams' disease) is easily diagnosticated if one merely takes care to count the pulse (see *Arhythmia Auriculoventricular Dissociation*). A subsequent thorough general examination and, if need be, a good polygraphic tracing will eliminate all doubt

5. Chloroformic syncope is, as is well known, a dangerous manifestation. Following are the warning signs of this condition, as recalled by Desfosses:

(*a*) Respiration. Arrest of respiration occurring together with pallor of the face.

(*b*) Facial appearance: "If the face is seen suddenly to become blanched or dusky, and the pupil to dilate, this means that the respiration, or perhaps the pulse, has just stopped; the result is 'white syncope' The patient is in extreme danger."

(*c*) Condition of the pulse· "If the pulse stops, there is present cardiac syncope, a very serious or 'white syncope'; but as a rule the arrest of respiration will already have served to warn the careful anesthetist."

(*d*) Examination of the eye· "If the pupil, after having been contracted, suddenly dilates, the corneal reflex should be tested at

once; if present, vomiting and return to consciousness are to be apprehended; if not present, a severe syncopal attack is to be feared."

6. All weakening, exhausting, debilitating illnesses lead to a manifest predisposition to faintness, which may pass into actual syncope. The mere change from the horizontal to the vertical position is sufficient, in many convalescents, to bring on a lipothymic attack Sphygmomanometric study of these cases reveals in this connection a considerable degree of tachycardia with markedly low blood pressure. As is well known, in such cases a post-infectious adrenal insufficiency, with low pressure, asthenia, and Sergent's white line, is nearly always found. This seems to be the case, in particular, in attacks of *pernicious malarial fever* (of the syncopal type).

7. Cases of syncope in the presence of extensive pleural effusion, with large areas of flatness, have been reported. "Do not wait till the patient faints to tap," wrote Trousseau. Doubtless it is through having followed this rule that the author has never witnessed syncope in these cases, neither spontaneously nor during the process of tapping

8. Any extensive hemorrhage, *e g.*, the intestinal hemorrhage of typhoid fever, the intraperitoneal hemorrhage of extra-uterine pregnancy, postpartum hemorrhage, internal hemorrhage following wounds of the chest or abdomen, or an uncontrolled external hemorrhage, bring on a syncopal condition which may pass into fatal syncope.

Syncopal states are, as a rule, easily distinguished, as was previously pointed out, from "syncopomorphic" hysterical seizures by their shorter duration (hysterical coma persists for minutes or hours; syncope only for seconds), the existence of an actual provocative cause, and especially, the *observation* of actual cardiac and vasomotor disturbances, such as slowing or suppression of the pulse, pallor, cold sweat, cooling of the tissues, etc.

# FEVER. [Febris, *from* φεβομαι, *to tremble.*]

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In practice, the words "fever," "pyrexia," and "hyperthermia" are often used indiscriminately, without marked disadvantage.

A patient is spoken of as having "fever" when his temperature is continuously above the normal.

The rectum and the floor of the mouth are the points of election for taking the so-called *"central"* or *"internal" temperature.* In adults the normal internal temperature ranges between 37° and 37.6° C. (98.6° and 99.68° F.), the physiologic oscillations of temperature during the 24 hours, and the difference between the minimal or morning and the maximal or evening temperature being sometimes as great as 0.5 to 0.6° C.

The axilla and the inguinal fold are the points of election for taking the so-called *"peripheral"* or *"superficial" temperature.* In adults the normal superficial temperature ranges between 36.4° and 37° C. (97.52° and 98.6° F.), with diurnal oscillations of 0.5 to 0.6° C. There is thus a mean interval of 0.5° C. between the internal and superficial temperatures. The sources of error, however, *viz.*, sweating, cooling of the surface, and faulty mode of application of the thermometer, are much more pronounced over the surface.

Preference should, therefore, be given wherever possible to the *internal temperature.* Ordinarily the internal, rectal, and oral temperatures are the same; but sometimes they are markedly different; it should be borne in mind that a local inflammation or hyperemia, as in proctitis, hemorrhoids, high portal pressure, etc., may result in a localized hyperthermia unaccompanied by true fever (*i.e.*, there is no actual pyrexia). The author has seen patients considered febrile and kept in bed for weeks or even months because of a rectal temperature persisting in the neighborhood of 38° C. (100.4° F.), but having no true fever, as was later proved by regular and careful notation of an absolutely normal buccal temperature

(37° to 37.4° C.—98 6° to 99.32° F.), a pulse rate of 60 to 72, and the absence of all symptoms, in spite of persistence of the rectal temperature in the vicinity of 38° C. All these were cases of proctitis, hemorrhoids, portal hypertension, or congestion of the liver. Sometimes repeated introduction of the thermometer, two or more times a day, or the local use of irritant antiseptics seem to be the exciting cause of the local irritation.

In a general way, as is well known, *fever is an indication of the*

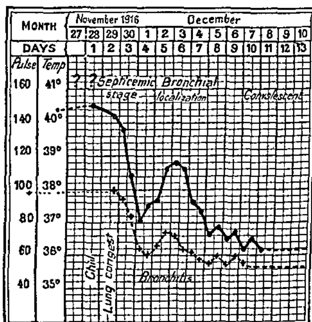



Fig 685.—Respiratory type of influenza.

*presence of infection.* Clinically, the equation, fever = infection, is justified in 19 cases out of 20. There remains, however, a small percentage of non-infectious fevers, to be briefly discussed later. To review all the causes of fever would thus entail a tiresome enumeration of all the infections, with the further addition of a few non-infectious forms of pyrexia

As a matter of fact, the solution of the clinical problem of fever is sometimes immediately manifest; erysipelas, herpes, the eruptive fevers, etc, become plainly apparent sooner or later.

In other instances the cause remains for a long time, if not permanently, obscure, and for a solution of the problem application of the most recent technical procedures is required.

From the exclusive standpoint of practical diagnosis, a useful clinical division of fever is that into:

**Fevers of short duration**, lasting altogether not over two weeks.

**Fevers of long duration**, persisting over two weeks without descending to normal

**Intermittent fevers**, or recurring fevers, made up of variable periods of pyrexia separated by intervals of apyrexia.

Little need be said concerning the **fevers of short duration**, since in these cases either the diagnosis becomes more or less

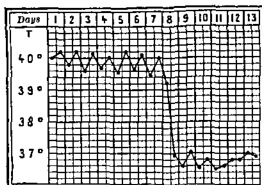



Fig 686—Frank pneumonia in an adult.

plain sooner or later, or, if the cause remains obscure (as is often the case), more or less prompt recovery occurs in any case, thus settling the main practical question of import to the patient as well as to the physician

In this group of fevers are encountered

The eruptive fevers or exanthemata Measles, scarlet fever, rubella, etc, and diphtheria.

Common or specific infections of the respiratory tract. Catarrhal conditions, acute bronchitis, sore throat, pharyngitis, pneumonia, bronchopneumonia, etc ; also influenza, etc

The ordinary gastro-intestinal infections: Febrile gastric upsets, acute gastro-enteritis, appendicitis, etc

Acute infections of the various other systems and structures· Acute arthritis, lymphangitis, pelvic infections, sinusitis, erysipelas, poliomyelitis, etc

In truth, most of these fevers of short duration are actually of unknown etiology and origin, or at least are "non-specific," and many of the terms applied to them, such as cold, grippe, influenza, febrile pains in the limbs, ephemeral fever, rheumatoid fever, etc., are none other than makeshift "clinical labels" applied to *"cryptogenic fevers,"* generally mild and of brief duration

Further, some of these conditions, persisting for unduly long periods, may pass into the group of the fevers of long duration, to be next considered, and conversely, a few of the clinical states ordinarily attended with fever of long duration may be cut short in some unusual way and fall into the present group The possible occurrence of such exceptional cases should be kept in mind For practical purposes the general division given above nonetheless remains of marked clinical service

**The fevers of long duration** are attended with a more urgent *need of proper diagnosis because in them prompt* recovery, which would quickly solve the clinical problem in spite of the physician's doubt, fails to occur

In 90 per cent of cases the underlying condition is either tuberculosis, typhoid fever, septicemia, or deep-seated suppuration.

The remaining 10 per cent. of cases refer to a wide variety *of conditions, including rheumatic fever, influenza,* meningitis, chronic appendicitis, leukemia, syphilis, cancer, etc

Cabot's statistics, referring to 784 febrile cases recorded at the Massachusetts General Hospital, are as follows:

| | |
|---|---|
| Typhoid fever | 586 |
| Sepsis | 70 |
| Tuberculosis | 54 |
| | 710 (90 per cent.) |
| Meningitis | 27 |
| Influenza | 10 |
| Acute rheumatism | 9 |
| Leukemia | 5 |
| Cancer | 2 |
| Syphilis | 2 |
| Trichiniasis | 2 |
| Cirrhosis | 2 |
| Gonorrhea | 2 |
| Scattering | 11 |
| | 74 (10 per cent.) |

These statistical results manifestly do not in the least represent those of private practice; the disproportionately great number of typhoid cases is accounted for by the special concentration of these

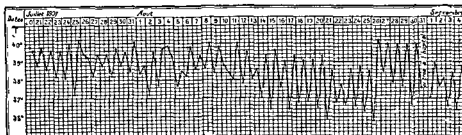



Fig 687—Temperature chart in a case of acute miliary tuberculosis with onset suggesting typhoid fever (*Letulle* and *Debré*).

cases in the Massachusetts General Hospital. In private practice tuberculosis, deep-seated sepsis, and the indefinite infections labelled

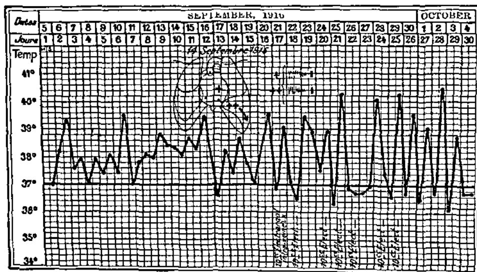



Fig 688—Infectious pericarditis.

influenza and rheumatism greatly exceed typhoid fever. Yet, on the whole, in private practice as in the statistics above presented, it may be said that 98 per cent. of the cases of prolonged fever fall, in the order of frequency, under the five following headings:

*Tuberculosis, sepsis of visceral origin, influenza, rheumatism, and typhoid fever.*

In *tuberculosis,* certain localizations of the infection are practically obvious, *e.g.,* tuberculosis of the bones and joints, lymphatics, peritoneum, meninges, reproductive organs, and pleura. It is particularly the pulmonary and the renal forms that may remain latent for a rather long period. The diagnostic means at the physician's disposal in modern special procedures are known to all: Auscultation, fluoroscopy, inoscopy, inoculation, etc.

Among the *septic conditions* one should think:

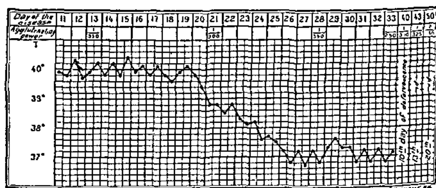



Fig 689—Typhoid fever, with recovery (*Widal* and *Sicard*)

1. Of *infectious vegetative endocarditis,* malignant and septic, very insidious, but revealed to a certainty by careful auscultation, by the characteristic temperature curve (the large oscillations of septicemia), and sometimes by complications (embolism).

2. Of *puerperal* and *post-puerperal infections,* generally obvious

3. Of *visceral infections, viz.,* in the order of frequency:

Appendicular and peri-appendicular infections

Cystic (gall-bladder), pericystic, and hepatic infections.

Urinary infections, renal and perirenal.

Genital infections, pelvic, prostatic, etc.

Gastrointestinal infections.

4. Of *pleural infections.*

5. Of *lymphatic infections,* lymphangitis, erysipelas, or abscesses

Deep-seated visceral abscesses, particularly those of the kidney and liver, are at times the hardest to detect because local symptoms

are often absent. All the resources of modern clinical investigation are to be availed of in such cases. It is in respect of these cases that the author has committed and seen committed the greatest

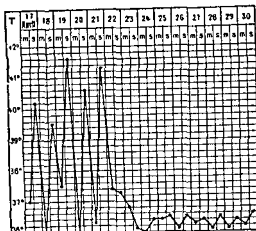



Fig 690 — Malaria. Intermittent fever of quotidian type (*Laveran*).

mistakes in diagnosis The possibility of these conditions should always be thought of in prolonged "cryptogenic" fever

In *influenza* with protracted fever, the chief problem, on the

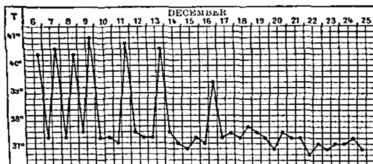



Fig. 691 — Malaria. Intermittent fever of quotidian type, then tertian, and finally quartan (*Laveran*)

whole, is to ascertain whether a threatening tuberculous infection is not concealed behind the screen of the acute infection, and if some deep-seated complication (liver, kidney, etc.) has not stepped in and changed the usual course of the disease. The diagnostic problem merges with that above referred to.

In *acute articular rheumatism* and the *post-infectious rheumatic states,* the joint involvement and the history generally render the diagnosis obvious.

The diagnosis of *typhoid fever, the paratyphoid fevers, and typhobacillosis* is founded on the frequently typical temperature curve, the sometimes very definite clinical picture, and especially on the modern methods of blood examination—serum diagnosis, blood cultures, and inoscopy.

Many cases of prolonged, obstinate *febricula* in childhood and even in adults, with temperatures of 36° to 37° C (96.8° to 98.6° F.) in the morning and 37.5° to 38.2° (99.5° to 100.76° F.) in the evening—accompanied at the latter period by slight malaise—wrongly ascribed to tuberculosis, are undoubtedly of *pharyngeal origin.*

* * *

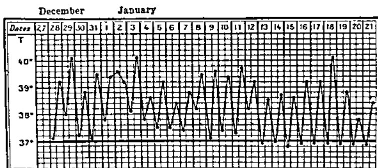
The above mentioned febrile affections make up, as already pointed out, 98 per cent. of all cases of prolonged fever. The remaining 2 per cent comprise a great variety of disorders, such as secondary syphilis (always to be kept in mind), meningitis (soon revealed by headache, Kernig's sign, etc ) and meningococcal conditions (often associated with herpes), the leukemias, rapidly growing malignant tumors (particularly cancer of the liver), Hodgkin's disease, Malta fever, etc.

Mention should also be made of certain exceptional and generally obvious conditions, such as wounds and diseases of the brain (cerebral hemorrhage, acute delirium, tumors, and skull fracture), belladonna poisoning, and toxic gas poisoning.

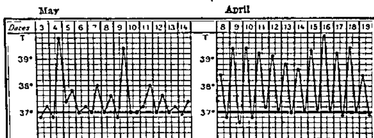
The majority of these clinical conditions are, as may be noticed, non-infectious

Along with this group may be cited the highly characteristic pyrexia of exophthalmic goiter, always of moderate degree, *viz.*, 38° C. (99.5° F.) or below, accompanied by a frequent pulse, generally reaching its maximum in the morning—as does likewise the pulse rate—and its minimum in the evening. With this form of pyrexia may, with Léopold-Lévi, be contrasted the customary hypothermia of cases of thyroid insufficiency, both these conditions being associated, moreover, with the usual concomitant dis-

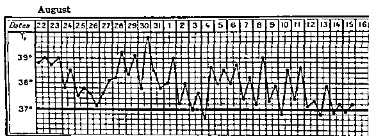
turbances of disordered thyroid function, *viz.*, circulatory disturbances (vasomotor or congestive), secretory disorders (hyperidrosis), and sensory symptoms (itching, burning sensations, etc.).




Intermittent fever of hepatic origin (suppurative angiocholitis).




Bilio-septic fever. Reversed type of intermittent hepatic fever.




Fever in relapsing jaundice (Weil Mathieu's disease)

Fig 692—Intermittent types of fever of hepatic origin (*Lereboullet*).

This type of hyperthermia may occur in nervous, neuroarthritic patients, during rapid growth, in persistent juvenility, in the various Basedow disorders, in thyro-testicular cases, at all periods of the reproductive life in the female, and in some high pressure cases.

## FEVERS OF LONG DURATION.

| Causes. | Predisposing Factors. | Onset. | Localization. | Characteristic Symptoms. | Blood Examination. | Special Tests |
|---|---|---|---|---|---|---|
| **Septic states.** | Low vitality. | **Rapid.** | Infective focus Heart | Large temperature oscillations Septic state Local evidences. | Polymorphonuclear leucocytosis Sometimes pathogenic germs in blood culture | |
| **Tuberculosis.** | Poverty. Overwork. Poor nutrition. Contagion. | **Slow.** | Lungs Pleuræ Bones Lymph-nodes | Irregular, erratic fever. Local evidences Auscultatory and fluoroscopic indications | Not characteristic. Lymphocytosis | Intradermal test. Examination for tubercle bacilli. |
| **Typhoid fever.** | August September October. Contaminated water or milk. Typhoid bacillus carriers. | **Gradual.** | Peyer's patches | Continuous fever. Rose spots Enlarged spleen Typhoid state, etc | Leucopenia. Agglutination test. Blood culture. | |
| **Influenza.** | Epidemic. | **Sudden.** | Respiratory tract. | Headache, backache, influenzal tongue, respiratory catarrh. | Leucocytosis. | |
| **Infectious arthritis (rheumatism).** | Cold and dampness | **Rapid.** | Joints (sometimes pleuræ and endocardium). | Signs of arthritis, sometimes with secondary involvements of endocardium and pleuræ | Moderate leucocytosis | |

| | | | | | | |
|---|---|---|---|---|---|---|
| **Meningitis.** | Nervous excitability | **Rapid.** | Nervous system. | Headache Kernig's sign<br>Condition of cerebrospinal fluid Cultures<br>Stupor Delirium. | Leucocytosis. | Examination of cerebrospinal fluid (lymphocytosis or leucocytosis). |
| **Leukemia.** | ? | **Slow.** | Spleen<br>Lymph-glands<br>Blood | Enlarged spleen<br>Enlarged lymph glands | *Leukemia* (*see* Examination of Blood). | |
| **Cancer.** | Heredity ?<br>Local irritation | **Slow.** | *Stomach*<br>Liver<br>Intestines, etc | Leukemia<br>Tumor of some organ<br>Progressive cachexia | Anemia, sometimes of pernicious type | |
| **Syphilis.** | Exposure | **Gradual.** | Skin Mucous membranes<br>Lymph-nodes<br>Bones Viscera, etc | Local evidences<br>Mucous patches, enlarged glands, eruptions, headache, etc | Wassermann reaction | |
| **Gonorrhea.** | Exposure. | **Rapid.** | Urethra and bladder<br>Prostate. | Gonorrhea | Gonococcemia (rare) | Examination for gonococci |

This fever of neuropathic origin may be compared with the so-called purely "nervous," "hysterical," or "psychic" fevers, our knowledge of which is as yet very deficient. How shall one account for the "admission fever" noticed when a patient enters a hospital, apparently a very real pyrexia, and the "Sunday fever," observed when visitors are admitted? In truth, many different factors may be operative under these circumstances.

**Intermittent Fevers.**—In practice this group of fevers is represented in our climes by *malaria*, detected from the history (former residence in a malarial district), the intermittent type of the fever, the enlarged spleen, and examination of the blood for the causal parasite.

*Relapsing fever* is very exceptional in temperate climates.

It should not be overlooked, however, that this intermittent or recurrent form of fever may be caused by non-specific, non-exotic infections, foremost among which are:

*Hepatic intermittent fever* (bilio-septic fever), especially frequent in angiocholitis and quite precisely reproducing the appearances of malarial intermittent fever, sometimes even to the extent of showing a maximum of temperature in the morning, constituting a "reversed" type of fever (Gilbert and Lereboullet). The fever may be remittent or even at times continuous in type (see page 937).

*Urinary intermittent fever* (uroseptic fever), especially common in pyelonephritis, involves precisely the same considerations.

Yet, while these infections may present the appearances of an attack of sudden onset, running a more or less rapid course, well maintained and with typical recurrences, they never exhibit the characteristic feature of regular intermittence shown in the true malarial paroxysms.

* * *

In conclusion, it may be well to recall, with Edmond Lesné, the following point of practical importance, *viz.*, that "THE INTERNAL TEMPERATURE OF THE BODY MAY BE CAUSED TO RISE BY ONE OF A NUMBER OF DIFFERENT FACTORS," which may be enumerated thus:

(1) *Elevation of the external temperature;* (2) *tetanic muscular contractions;* (3) *lesions of the nerve centers;* (4) *infections.* When present in combination, these several factors bring about the so-called "*hyperthermic fevers,*" e.g., in meningitis attended with convulsions.

## FREQUENT PULSE.

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Tachycardia, or frequent pulse, is marked by a more or less pronounced and persistent acceleration of the heart beats Normally, in sitting or recumbent subjects, the pulse frequency ranges from 60 to 80 in different individuals, at different times of the day, in accordance with the intervals before or after a meal, etc For practical purposes, tachycardia or frequent pulse can hardly be said to exist unless the rate exceeds 90; it may attain and even exceed 200.

Accelerated heart action is a commonly and easily observed condition. Its clinical meaning is sometimes obvious and of slight import; under other circumstances, however, its significance is so great as to make of it a separate morbid entity, *viz.*, paroxysmal tachycardia, a condition which has excited wide interest among modern clinicians Rather frequently the proper interpretation of a frequent pulse proves a matter of considerable difficulty.

For practical purposes, the *frequent pulse* may be said to occur in three separate modalities which lend themselves to rapid differentiation · 1. More or less permanent tachycardia. 2 Attacks of paroxysmal tachycardia 3. Temporary, accidental attacks of tachycardia.

I. **More or Less Permanent Tachycardia.**—This is met with under two groups of circumstances, sometimes clearly defined and separate, at others in combination.

(*a*) In the presence of a *recognized heart lesion*, such as myocarditis, pericarditis, and valvular disorders, especially aortic.

(*b*) In the absence of any *recognized heart lesion:*

(1) Graves's disease (exophthalmic goiter).

(2) Tachycardic neuroses

(*a*) **The frequent pulse occurring as a symptom of organic heart lesions should generally** *be* **attributed to a more or less pronounced weakness of the myocardium.** The reduction in the

tachycardia following rest, good hygiene, and digitalis when properly administered constitutes evidence of the truth of this supposition. The results of the static and dynamic tests of the circulation may be said to afford also an experimental demonstration. This type of frequent pulse is met with in association with myocarditis (particularly post-infectious), pericarditis, and endocarditis, especially aortic. The diagnosis should be based on: 1. The presence of the characteristic clinical signs of these conditions 2. The results of the special test of the circulation already referred to 3 The effects of treatment, as by rest, diet, and digitalis What remains in the way of a

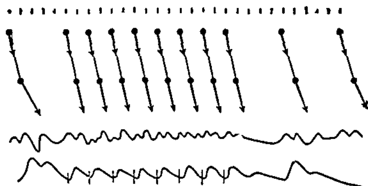


Fig 693—Diagram representing a brief attack of paroxysmal tachycardia consisting of 8 auricular extra-systoles With each auricular impulse there corresponds a ventricular contraction Note the abrupt onset and termination of the attack and the abnormal prolongation of the terminal pause

tachycardia under these circumstances constitutes an index either of irreducible myocardial weakness or of a concomitant tachycardic neurosis

It should be borne in mind, moreover, that in some individuals the most varied forms of tachycardia may be observed occurring in succession, *viz*, tachy-arhythmia, paroxysmal tachycardia, persistent tachycardia of the Graves's disease type, premature contractions, etc. These cases are exceptional, but many instances of them have nevertheless been recorded

(*b*) "Yet," as Gallavardin correctly writes, "it must be confessed that bordering on these sinus tachycardias of known origin there occurs a vast group of *tachycardias of poorly determined*

*origin*, generally appearing in young or adult subjects, and concerning which comparatively little is known. Some of these cases are unquestionably to be ascribed to a *fruste Graves's disease*, for, in spite of the absence of any ocular sign of this disorder, careful examination often elicits a small nodular goiter or merely a neck slightly broadened at its base, with slight diffuse enlargement of the thyroid lobe (usually the right). In other instances, however, nothing is found upon examination of the thyroid These cases are well known, and no physician exists who has not seen some of them; it would seem well to group them provisionally under the generic term *tachycardic neuroses*. The accelerated heart

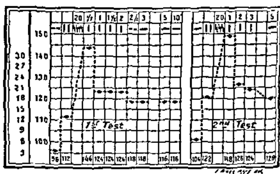



Fig. 694—Heart weakness (H, 1893, 165 cm, 60 kilogr)
Experimentally induced tachycardia

action, an aggregate of symptoms of disturbed function, sometimes highly distressing (palpitations, breathlessness on exertion, and various painful reactions), and lastly, the absence of any noticeable cardiac lesion constitute the basic symptomatic triad which may serve as a foundation for all the cases of this kind."

The frequent pulse of Graves's disease origin is obvious from the recognized symptomatic accompaniments of this condition, *viz*, tachycardia, exophthalmos, hyperthyroidism, and secondarily, tremor and vasomotor disturbances.

The tachycardias of cryptogenic origin (tachycardic neuroses) already mentioned are, aside from the absence of exophthalmus and of hyperthyroidism, identical with the tachycardia of Graves's disease.[1]

[1] See MARTINET: "War and the Cardiac Neuroses" (*Presse méd*, Nov. 5, 1915)

Both forms are enhanced by exercise, are only rarely accompanied by dyspnea or premature contractions, and are but slightly or not at all influenced by compression of the eyeballs or treatment with digitalis; a moderate elevation blood-pressure is nearly constant in these cases A very common, if not constant, phenomenon should also be referred to, *viz.*, inversion of the temperature and pulse rate, the pulse frequency diminishing while the body temperature ascends. Gallavardin has made a special study of this phenomenon, and the author of this work has frequently had occasion to see it. Tachycardia of this type

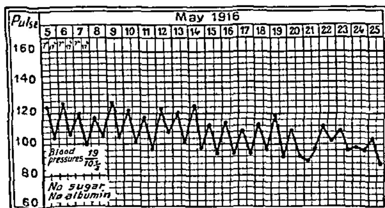



Fig 695—Tachycardic neurosis (Case V, 458, H., 1896, 178 cm; 65 kilogr)

Note the rise in heart-rate each morning

is at its maximum in the morning and its minimum in the evening

The author freely agrees with the conclusions expressed by Gallavardin:

"In the two instances (fruste Graves's disease and tachycardic neuroses), the clinical syndrome is really the same There is the same variability in the heart acceleration, the same morning type of tachycardia, the same change in the temperature and pulse rate cycle, and the same clinical features, with continuous or temporary tachycardia The condition may always be summarized as an *intense excitation of the sympathetic*, with not only a cardio-accelerator, but also a vasoconstrictor and blood-pressure-raising and even

a thermic action. One must actually make bold to state that *these two varieties of patients differ only as regards the neck enlargement.*

"Hence two modes of interpretation are allowable. *Is one dealing with two thyroid disorders,* the one with a manifest goiter, the other with some concealed change in the thyroid (small adenomas, defects in the internal secretion), or merely with two pathogenetically distinct *syndromes of sympathetic excitation, the one of thyroid origin and the other of unknown causation?* While the first of these two theories seems the most likely and many authors have already spoken of cases of fruste Basedow's disease, without goiter, and with tachycardia alone present, it will be wise, before reaching a conclusion, to await positive proof."

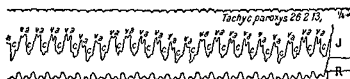



Fig 696—Paroxysmal tachycardia (*Routier*).

In this group of cases belongs the *continuous post-emotional* form of frequent pulse.

II. **Attacks of paroxysmal tachycardia,** coming on, as the term implies, in paroxysmal outbursts, starting and ending abruptly The very nature of the attacks of tachycardia, their marked clinical autonomy, and their rather clearly elucidated pathogenesis, as developed in late years, which makes of them *actual extra-systolic seizures brought* on by the coming into play of an abnormal center of cardiac motor excitation—all these facts contribute to their formation into a special group of *tachycardias* which are *really abnormal, arhythmic,* and, as a rule, readily distinguished.

The diagnosis of this disorder is rather easy. It may be put down as a definite rule that any *tachycardia exceeding* 110, of abrupt onset, unaccompanied by exophthalmic goiter, not coming on in association with some febrile disorder, and *the rate of which is not greatly altered by passage from the recumbent to the upright posture,* is a paroxysmal tachycardia. The only difficulty arises in subjects seen for the first time, whose history is not

known, and who, in conjunction with tachycardia of varying degree, present obvious signs of heart failure, such as dilatation of the heart, edema of the lungs, congestion of the liver and enlarged spleen, reduced urinary output, and edema. In such cases it may

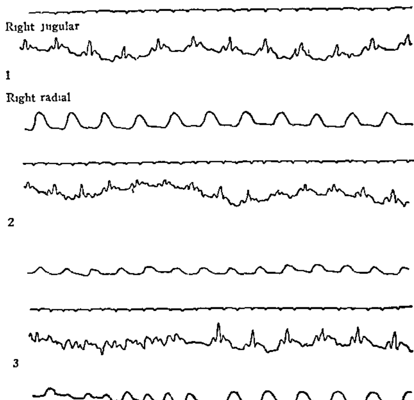



Fig 697—Case 1219, Mar 18, 1915 (F, 1867, 158 cm, 48 kilogr)

Attack of paroxysmal tachycardia of a few minutes' duration (*1*, beginning, *2*, middle, *3*, end of attack) in an improved case of exophthalmic goiter at the menopause Note the abrupt onset, abrupt termination, and clearly extrasystolic terminal period

be difficult to ascertain whether the paroxysmal tachycardia was the initial morbid manifestation or, on the other hand, the tachyarhythmia witnessed is not a secondary result of the impaired heart action The sudden onset, accurate graphic records, and the therapeutic test with digitalis will settle the question

III. **Temporary, accidental attacks of tachycardia** are those in which there is simply acceleration of a heart beating in normal rhythm. The most common and manifest causes of such attacks may be briefly recalled as follows:

(*a*) **Fever.**—Pulse acceleration and hyperthermia are the two characteristic, clinical, and essential features of fever. As is well known, in some kinds of fever, as in meningitis, peritonitis, etc., there may be noted a discrepancy between the degree of hyperthermia and the increased pulse rate, and such a discrepancy is in itself a clinical indication of by no means negligible value.

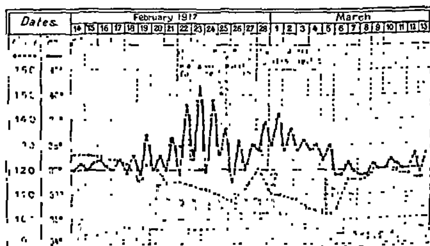



Fig 698—Case V, 750 (H, 1897, 168 cm). Post-infectious pericarditis with extensive effusion

It should also be remembered, as will be mentioned again later, that hyperthermia may exert a sedative, slowing action on certain forms of continuous tachycardia (Graves's disease, tachycardic attacks). In one case of pericarditis with extensive effusion (in which puncture was carried out) which the author had occasion to observe closely for a long period, two successive attacks of acute suppurative otitis media caused, in addition to marked fever (39°-40° C.), an equally pronounced slowing of the pulse, which was reduced in the two attacks from 124 to 104 during the period of fever, rising again to 120 or above in the interval and after the second attack. Herein lies a rather odd application of the well-known aphorism: *similia similibus curantur.*

(*b*) **Exertion.**—That the pulse rate rises during exertion, and ascends to a degree varying with the extent of the exertion and its duration, is a matter of common observation Upon the basis of this fact the author has devised a test of induced tachycardia brought on by a standard amount of exertion, which affords some measure of information concerning the reserve power of any given heart. In a general way it may be asserted that after a medium amount of exertion the acceleration of the pulse as compared to the rate while at rest is less, and the return to the *status quo ante* after cessation of exertion more rapid, according to the amount of reserve power possessed by the heart.

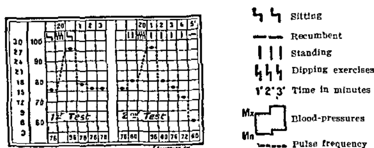



Fig 699—Normal individual (H, 1884, 149 cm, 46 5 kilogr.). Experimental tachycardia, after exercises

(*c*) **Posture.**—Acceleration of the pulse due to passage from the recumbent to the upright posture, or *orthostatic tachycardia*, is likewise a well-known event Prevel seems to have found out an essential factor in this form of tachycardia, ascribing it largely to visceroptosis, making of it an abdominocardiac reflex, and demonstrating that it can be reduced or eliminated by the wearing of an abdominal support.

(*d*) **Emotion**—The well-known "doctor's pulse," an emotional tachycardia which the physician regularly observes, especially at his earlier visits, should not lead him into error, it generally subsides after he has been talking with the patient for a few minutes Pulse acceleration is one of the most constant somatic signs of the emotional syndrome. There actually exists an emotional tachycardic constitution—a true tachycardic neurosis

(*e*) **Pain.**—Many forms of *visceralgia* bring on an evanescent reflex tachycardia, the source of which may be as likely found in the uterus as in the pleura.

(*f*) Certain kinds of **intoxication**, among the foremost of which should be placed *coffeeism* and *teaism, caffeinism* (caffeine) and *theobrominism* (theobromine). At first the heart acceleration is transitory and present only during the period of intoxication; later, however, there may be seen to develop an actual *tachycardic neurosis.* Hence the rule, which is, on the whole, frequently justified, *of forbidding the use of tea and coffee in persons with "excited" hearts.*

Lastly, it may be noted that while the heart-rate is in normal subjects entirely independent of cerebral volition, 15 authentic cases have been recorded in medical literature of individuals who could at will markedly increase the frequency of their heart beats.

## TACHYCARDIA

I **Paroxysmal.**

II **Temporary. Accidental** Easily referred to the following causes

1 Fever Febrile pulse acceleration
2. Exertion· Pulse acceleration on exertion
3. Posture. Orthostatic pulse acceleration { Experimental pulse acceleration (cardiac functional test).
4 Emotion· Emotional pulse acceleration
5 Pain Algic pulse acceleration
6. Intoxications· Toxic pulse acceleration (tea, coffee, caffeine, kola, theobromine)

III **Continuous.**

A. *In the presence of a recognized heart lesion: Frequently a reactive effect of heart weakness or failure*
B In the absence of any recognized heart lesion.
B[1]. Graves's disease
(*a*) Cardinal symptoms Accelerated pulse, exophthalmos, hyperthyroidia (goiter).
(*b*) Accessory symptoms Tremor, vasomotor disturbances, excessive nervousness
B[2]. Tachycardic neuroses Features same as those of Graves's disease, with the exception of the goiter and sometimes the exophthalmos "These two kinds of disorder differ only in the appearance of the neck" (Gallavardin)

# GENITAL ULCERATIONS.

Recognition of the cause of ulcers on the genitals is, on account of the frequent presence of syphilis, of great clinical importance. A condensed tabular presentation of the main facts in this connection seems, therefore, appropriate.

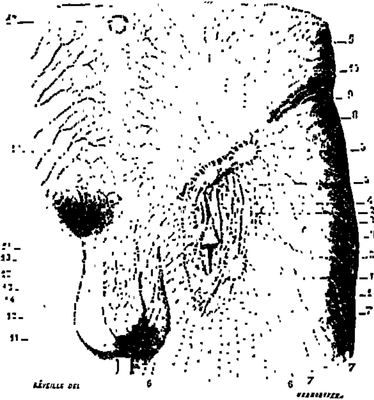



Fig 700—Inguinal lymph-nodes, with the afferent and efferent lymphatic vessels (*Sappey*).

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Fig 701—Hard chancre of the penis (*Sabouraud*).

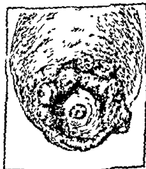


Fig. 702—Chancroidal pus, stained with methylene blue



Fig 703—Scrapings from hard chancre (Giemsa stain)





Figs 704 and 705—*Unusual* papulohypertrophic chancroids of the balanopreputial region (*Marcel Ferrand*)

## GENITAL ULCERATIONS

| MACROSCOPIC APPEARANCE INCUBATION PERIOD | GLANDULAR ENLARGEMENTS | ASSOCIATED CLINICAL EVIDENCES COURSE | VARIOUS TESTS MICROSCOPIC EXAMINATION |
|---|---|---|---|
| **Hard Chancre.** | | | |
| 1 Flat ulcer, non-suppurative, *generally* single<br>2 Resting on a plate of cartilaginous induration (see Fig 701)<br>Fourteen to twenty-eight days after coitus. | In contrast to chancroidal glandular enlargement, the non-inflammatory nature of the glandular enlargements attending chancre is to be noted<br>1 **Ricord's Pleiades**<br>Secondary syphilitic multiple glandular enlargement<br>Multiple, elastic, movable, and painless glands<br>2 **Indicating** (primarily enlarged) **gland,** or satellite of the chancre, somewhat larger | Later on, roseolar eruption, mucous patches, patchy alopecia, etc | Later on, the secondary stage<br>Positive Wassermann test<br>Negative reinoculation in the same subject upon insertion by puncture of material from the initial lesion.<br>Ultra-microscopic examination.<br>Giemsa stain.<br>Schaudinn's pale spirochete (see Fig 703) |

Apart from the initial specific lesion, **syphilis** may be manifested on the glans penis in several other ways

1 **Papules** Secondary papular syphilides, varying in number (Recognized from the history, absence of lymphatic reaction, and the effects of specific treatment)

2 **Gummas** Rather rare, gummatous nodules undergoing absorption under treatment or terminating in vicious and deforming scars

3 **Chancriform gummas** Similar to the initial lesion (false reinfection, false syphilis redux, etc ), no roseolar eruption and no glandular enlargement

### Chancroid.

| | | | |
|---|---|---|---|
| 1 Vesiculo-ulcerations.<br>2 Then suppurating ulcers<br>3 Then multiple punched-out ulcers, suppurating freely, without induration (see Figs 704 and 705)<br>Appear four to eight days after coitus | 1. **Single bubo,** soft, painful, terminating in suppu-<br>2 | In some instances phagedenic tend- | Positive reinoculation in the same subject upon in- |

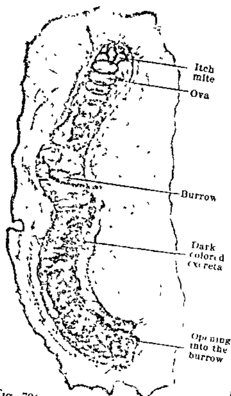
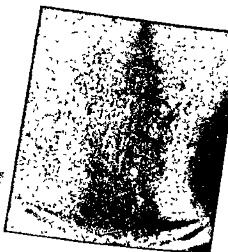
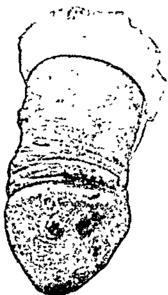
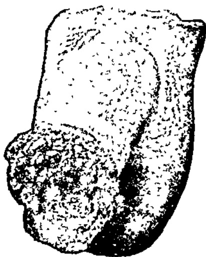


Fig 706 —A burrow in scabies (*Darier*)


Fig 707 —Deep-seated vulvar herpes (*Darier*)




Figs 708 and 709 —Vegetations on penis (*Musée de Saint-Louis*)

## GENITAL ULCERATIONS (*continued*).

| Macroscopic Appearance Incubation Period | Glandular Enlargements | Associated Clinical Evidences Course Various Tests. |
|---|---|---|
| | **Herpes.** | |
| 1. Vesico-pustules grouped together, then, 2. Superficial, circinate, polycyclic ulcerations One or two days after irritation or without appreciable cause. | Glandular reaction very slight or wanting | Sometimes recurrence *in situ* with discouraging obstinacy |
| | **Scabies.** | |
| 1. Round, red, flat, almost papular spots 2. Sometimes distinct burrows 3 Similar lesions on the prepuces, in the groins and axillæ, etc Characteristic feature of the acquisition of an itching affection by contact. | Glandular reaction very slight or wanting | 1 Itching in nocturnal paroxysms 2 Lesions due to scratching 3 Lesions at the points of election Groins, armpits, forearms, etc. 4 Characteristic burrows Itch parasites in the burrows (See Parasitology and Itching ) |
| | **Cauliflower or coxcomb genital vegetations** | |
| 1 More or less prominent papules ultimately forming, by proliferation 2 Excrescences, tumorlike masses, or vegetations, attended with a varying degree of suppuration Perhaps of gonorrheal origin | Glandular reaction very slight or wanting. | Persistent, rebellious, and recurrent owing to a manifest predisposition to epithelial proliferation |
| | **Genital diabetides** | |
| Nothing characteristic Oozing erosions and eczematoid balanitis | Glandular reaction very slight or wanting | Refractory to all measures other than antidiabetic treatment Glycosuria |
| | **Balanitis.** | |
| More or less copious and rebellious suppuration, most pronounced over the glans and prepuce | | To be examined for: Diabetes (see above) Mercurial poisoning. Potassium iodide poisoning Erosive circinate balanitis Vegetations, herpes, chancre, etc |

# GLANDULAR ENLARGEMENTS.

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**Adenitis** or adenopathy [ἀδήν *gland*] consists generally of a morbid enlargement of one or more lymphatic ganglia.

It is rather hard to define exactly at what degree of enlargement "adenopathy" begins.

Indeed, in stout individuals none of the lymph glands, even those superficially situated, are normally palpable; in very thin persons, on the contrary, they are easily palpable at certain points, *viz.*, in the inguinal and axillary regions, in no individuals are they normally distinguishable in regions other than those just mentioned.

The **diagnosis of glandular enlargements** is hardly open to error. *Lipomas* are of an altogether different, soft and lobulated consistency; *sebaceous cysts* (wens) are embedded in the skin proper, and their localization is usually quite different from that of glandular enlargements, ***actinomycotic skin infiltrations*** are intradermal in situation

Morbid enlargements of these lymphatic ganglia *generally* correspond to certain definite areas or anatomic regions.

Considering for a moment only the *readily and actually accessible glandular regions,* it may be considered a general rule that swollen glandular masses correspond to the several anatomic divisions of the body as follows

**Inguinal glands.**—These drain the reproductive organs, the lower extremity, and very exceptionally react to pelvic or abdominal affections

**Axillary glands.**—These are related to the thoracic wall, including the breast, and the upper extremity, and very exceptionally react to thoracic tumors.

**Post-cervical glands.**—These drain the mouth, throat, face, and cranium.

**Submaxillary glands**.—Related to the lower jaw.

**Supraclavicular glands**—These exceptionally exhibit metastasis from cancer of the stomach

It should be remembered, however, that the majority of lymphatic enlargements completely escape our examinations by reason of their deep situation in the body

Thus, abdominopelvic tumors and infections almost inevitably give rise to inaccessible *deep mesenteric and prevertebral glandular*

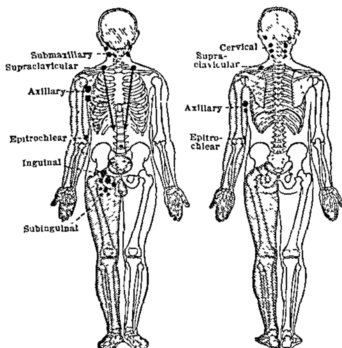



Fig 710—Superficial lymph-nodes and the related anatomic regions.

*swellings in the abdomen,* and only in exceptional instances to enlargement of the inguinal glands Tumors and infections in the chest almost inevitably give rise to inaccessible *tracheobronchial glandular enlargements,* and only exceptionally to enlargement of the cervical and axillary lymphatics

Such a condition is, on the whole, the rule, thus, cancer of the stomach, tuberculous peritonitis, infections of the biliary tract, gastric and duodenal ulcers, in fact, the great majority of abdominal disorders that are hard to diagnose are unaccompanied by any ap-

preciable enlargement of superficial lymphatic glands. Only exceptionally and in the terminal stage, and only in certain forms, are enlarged supraclavicular glands found in cancer of the stomach.

The tonsils, as is well known, like the follicles of the intestine, may and should be looked upon as actual submucous, pharyngeal and intestinal, lymphatic ganglia. The frequency with which they become diseased and the common occurrence of *sore throat* and of *intestinal folliculitis*, specific or non-specific, is also a matter of common observation Do these infections take place usually through the blood stream or through the alimentary tract? Do they constitute oftener a portal of entry or a focus of secondary involvement? An answer to this question is of marked theoretical as well as practical (therapeutic) importance; from our present exclusive standpoint of semeiology, however, it is of much less significance.

Finally, mention may be made of the *lymphadenomas*, or neoplastic enlargements of lymphatic foci normally not palpable.

In this work space will not permit of more than a review of certain common clinical observations susceptible to everyday application in the causal diagnosis of glandular enlargements.

In whatever region a **glandular enlargement** be situated, it may present itself to the examiner in one of the four following forms

(*a*) **Simple glandular hypertrophy** or **painless adenopathy** of intermediate extent and generally involving several lymph nodes. This includes the *syphilitic adenomas*, *lymphatic hypertrophies* with or without accompanying leukemia, *cold tuberculous glandular swellings*, and *lymphatic enlargements* which one is compelled to qualify as *cryptogenic*, since in last analysis their exact nature is beyond our ken, *e g.*, the lymphatic enlargements of *convalescence*, *of the "lymphatic temperament"* ("lymphatism"), etc

The **syphilitic gland enlargements** deserve especial mention both on account of their frequent occurrence and their diagnostic signification, sometimes practically pathognomonic.

*Glandular swelling is a constant appurtenance of the syphilitic chancre.* It involves either a single, large, hard, sluggish node, which never suppurates; or a group of nodes in the center of which is one larger than the rest.

*In the secondary stage, the lymph-glands become enlarged in all parts of the body,* including not only the glands corresponding to the lesions on the skin and mucous membranes in the secondary stage, but also those of other regions, the epitrochlear and post-cervical glands are of prime diagnostic import in this connection.

(*b*) **Inflammatory glandular hypertrophy or painful adenopathy,** involving one or more glands, with or without suppuration This is the ordinary type of *septic adenitis* following local infection· Cervical and submaxillary lymphatic enlargements, etc., in sore throat and infective disorders of the mouth or pharynx; enlarged inguinal and femoral glands in wounds and infections of the lower extremities and infective disorders of the genital organs (gonorrhea, balanoposthitis, and chancroid); enlargement of the axillary glands in infections of the upper extremities, and secondary lymphadenitis in boils or carbuncles in any portion of the body These disturbances may result in the production of glandular abscesses or, as is more common, undergo absorption

General infections of the type of "grippe" may cause temporary painful enlargement of "glands" that have been latent for a number of years

(*c*) **Caseous glandular enlargement, or gland softening** This is generally the result of *tuberculous lymphadenitis,* the usual precursor of the so-called "cold abscesses," which, if improperly treated, become adherent to and break through the skin, leaving the permanent, highly characteristic scars still rather frequently noticed in the cervical region

Adhesion of lymph-glands to the skin is met with particularly in the septic and tuberculous varieties of lymphadenitis

(*d*) **Hard, nodular, scirrhous glandular enlargements.**—These are usually the result of *neoplastic metastasis* from cancer, either manifest or latent; on the whole, they are generally *secondary malignant glandular enlargements* Some varieties are of great clinical import, *e g.*, the secondary enlargement of the axillary lymphatics in cancer of the breast and the rather exceptional supraclavicular enlargements in malignant growths of the stomach.

**Cervical lymphadenitis,** a very common disorder, and one readily observed in the exposed neck region, deserves especial mention. Of all gland swellings these are the most accessible, and they should be examined for as a routine.

**Submaxillary and post-maxillary lymphadenitis** is almost constant in children, being dependent upon one of the ordinary infections of the mouth or pharynx (sore throat, dental infection, etc), which few individuals escape.

**Lateral cervical adenitis** is also extremely common. The nasopharynx is the usual portal of entry to the infecting germ in these cases, and the tubercle bacillus is the commonest of the infecting germs The submaxillary and sternomastoid series of glands generally become involved at practically the same time All different varieties may be observed, from the *multiple slight enlargement* of several nodes (micropolyadenitis) to the *caseous lymphadenitis* terminating in cold abscess. *Scrofula* and *tuberculous infection* appear to be the commonest causes of these glandular affections

**Occipital adenitis,** manifest about the margins of the hairy scalp, on either side of the occiput, is encountered chiefly in the *presence of scalp infections (as in phthiriasis or impetigo)*; accordingly, it is seen more especially in children in the form of tender gland swellings which never undergo suppuration

**Syphilitic glandular enlargements** occur in the cervical region in two equally characteristic forms

(*a*) STERNOMASTOID ADENOPATHY IN CHANCRE OF THE TONSIL.— This occurs as an initial indication of primary general syphilitic glandular enlargement, represented by a single large node (*ganglion indicateur*), beneath and at the middle of the sternomastoid muscle, of the size of a walnut or hazelnut, and rendering the muscle tissue prominent above the surrounding surface, and a conjoint group or "pleiad" of smaller nodes underlying the whole neck region on the same side, hard, painless, and rolling beneath the finger. Careful inspection of the patient's throat will reveal a chancre of the tonsil on the same side, or, if it has already been absorbed, the patient will recall having had a tonsillitis on one side lasting a few weeks.

*Chancre of the tip of the tongue or of the lips* gives rise to enlargement of a suprahyoid node, "single, movable, and rolling

beneath the finger like a little rubber ball, the feel of which it exactly reproduces" (Sabouraud)

(*b*) SECONDARY SYPHILITIC ADENOPATHY is chiefly *post-cervical*, the nodes extending along the neck like a string of beads, movable, elastic, and practically insensitive. These should always be systematically examined for, and if they are found, the other signs, stigmata, and the history of the disease should likewise be sought.

**Neoplastic glandular enlargements** are secondary to cancer of the tongue or lips

Cancer of the base or lateral portions of the tongue reacts on the submaxillary and postmaxillary glands, and later on the sternomastoid group.

Cancer of the lips or of the tip of the tongue gives rise at first to suprahyoid glandular enlargement. Later, it extends to all the other lymph nodes in the region

These glandular enlargements are hard, nodular, and scirrhous, tending to become adherent and to infiltrate the surrounding tissues

The lymphadenosis of **Hodgkin's disease** generally appears first in the cervical region and is predominant in this location for a long time in the form of multiple, sometimes very large masses (varying in size from an almond to an orange), hard, insensitive, freely movable, not adherent to the skin, and never undergoing suppuration Examination of the blood shows a moderate leucocytosis with lymphocytosis (see below)

* * *

Special reference should be made to the **apparently primary tumor growths of the lymphatic and adenoid tissues.** These comprise the large group of the lymphomas, lymphadenomas, and lymphosarcomas, with or without leukemia.

The glandular enlargements are generally made up of hypertrophied nodes exhibiting structurally one of several different types

1 The first and commonest type is characterized by hyperplasia of lymphoid tissue similar to the normal tissue of the

lymph-nodes, *i.e.*, by proliferation of the lymphocytic cells (**lymphocytomatosis**).

2. A second, less common type is that which reproduces myeloid tissue, or the tissue of which bone marrow is composed, and is characterized by the presence of myelocytes and nucleated red cells (**myelomatosis**).

3. More rarely, the growths are made up of large mononuclear cells with basophilic non-granular protoplasm and clear nuclei (**macrolymphocytes or primitive cells**), like those seen in acute leukemia.

4. The last group brings together a number of very different kinds of tumors, all characterized by a special malignancy of growth (**malignant or atypical lymphadenosis**). It includes:

(*a*) **Lymphosarcoma**, consisting of sarcoma cells.

(*b*) **Granulomas**, the hybrid structure of which is characterized by a combination of lymphoid and myeloid hyperplasia with neoplastic and inflammatory proclivities.

**The condition of the blood reflects, as a rule, that of the blood-forming organs; hence the need of blood examination in the diagnosis of lymphatic adenopathies.**

*The ease with which this mode of examination may be carried out renders it accessible to the practitioner.*

A mere leucocyte count will show whether the case is one of leukemia.

Qualitative examination is, however, equally indispensable for revealing a lymphocytemia or a myelemia, which, even in the absence of leukemia, then becomes the main clinical feature.

A few stained, dry blood preparations will readily permit of ascertaining the leucocytic formula.

Yet the typical blood reaction may be lacking (though uncommonly), especially at first; in this event, removal of a bit of the glandular tissue for histologic study may be of great assistance.

**Clinically**, with the aid of the blood examination, one will be able to distinguish:

(*a*) **Hodgkin's disease** (lymphadenoma, aleukemic lymphadenosis, Trousseau's adenosis).

In this condition the glandular enlargement develops slowly, at the angle of the jaw, in the submaxillary region, or along the carotid chain of glands, often symmetrically; other glands then appear and fuse with the primary group, thus forming a mass which is sometimes of considerable size (up to that of a mandarin orange) This development takes place in the course of several months or even years The enlarged nodes are freely movable beneath the skin, and never undergo ulceration or suppuration.

Similar ganglionic masses occur in the axillæ and groins.

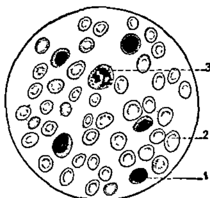



Fig 711—Blood in Hodgkin's disease *1* Lymphocyte. *2* Red cell *3* Polymorphonuclear leucocyte.

Hypertrophy of the spleen, liver, tonsils, testicles, etc, may likewise be observed

Blood Examination —Cell Count.—The red cells show a slight decrease, numbering 4 to 5 millions

The white cells do not show a leucocytosis, numbering 3000 to 5000

Or, there may be a moderate increase of the white cells—to about 25,000—constituting a *subleukemia.*

Differential Count.—Generally there is a lymphocythemia·

True lymphocytes 60 to 90 per cent.

Polymorphonuclear leucocytes· less than 10 per cent

Rarely, myelemia (presence of myelocytes or granular mononuclears and of nucleated red cells)

Rarely, an ordinary polymorphonuclear increase.

Rarely, again, the differential count may be normal.

The DIFFERENTIAL DIAGNOSIS of Hodgkin's disease involves especially the elimination of:

1. *Infectious glandular enlargements.*

In these there are either no blood changes or there is polymorphonuclear leucocytosis.

2. *Tuberculous lymphadenitis.*

In these cases either the blood formula remains practically normal or there may be a polymorphonuclear leucocytosis

(*b*) **Lymphatic leukemia**, the ordinary form of which is the splenoganglionic form, is characterized by gland enlargements,

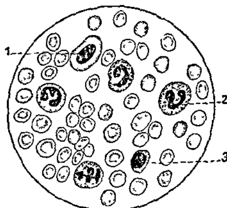



Fig 712.—Blood in tuberculous or other infectious glandular enlargement *1* Large mononuclear *2* Polymorphonuclear leucocyte. *3* Lymphocyte

either slowly or rapidly progressive, in the neck, the submaxillary and nuchal regions, the axillæ, and later in the inguinal regions.

Enlargement of the spleen is neither constant nor pronounced in these cases.

BLOOD EXAMINATION.—CELL COUNT.—The red cells show a decrease, frequently slight.

The white cells show a leucocytosis, often less marked than in myeloid leukemia—100,000 to 250,000, occasionally up to 900,000

DIFFERENTIAL COUNT.—True lymphocytes: 90 to 99 per cent.

Polymorphonuclear leucocytes: barely 10 per cent.

Eosinophiles: none.

(c) Myeloid leukemia, uncommonly.

Enlargement of the lymph-glands is uncommon in myeloid (spleno-myelogenous) leukemia, which is characterized mainly by enlarged spleen and liver, etc

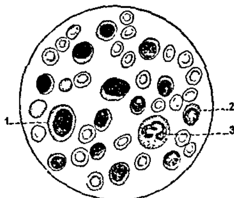



Fig 713—Lymphatic leukemia 1 Large lymphocyte. 2 Small lymphocyte 3 Polymorphonuclear leucocyte.

Blood Examination.—Cell Count.—The red cells show a marked reduction, to about 3 millions

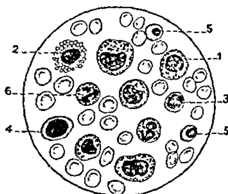



Fig 714—Myeloid leukemia 1 Neutrophilic myelocyte. 2 Eosinophilic myelocyte 3 Small lymphocyte 4 Large lymphocyte 5 Nucleated red cell 6 Polymorphonuclear neutrophilic leucocyte

The white cells are increased to about 300,000, up to 1 million

Differential Count.—The myelocytes or granular mononuclears predominate

Nucleated red cells are present in varying numbers.

(d) Acute leukemia.—The various groups of lymph-nodes, particularly the cervical and submaxillary, are involved, but

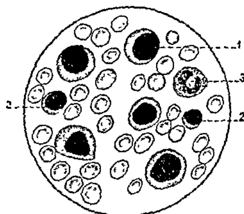



Fig 715—Acute leukemia *1.* Macrolymphocyte. *2.* True lymphocyte. *3* Polymorphonuclear leucocyte.

hardly reach the size of an almond. Enlargement of the spleen is slight.

There is tonsillar hypertrophy, suggesting an acute tonsillitis.

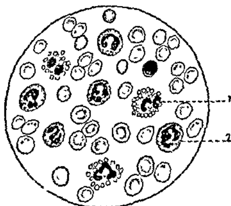



Fig 716—Lymphosarcoma *1* Eosinophile *2.* Polymorphonuclear neutrophile.

Hemorrhages, pronounced anemia, and fever.

BLOOD EXAMINATION.—CELL COUNT.—The red cells show a marked decrease.

The white cells are increased to 50,000 or 100,000; sometimes 200,000, or even 900,000

Differential Count.—Polymorphonuclears: below 10 per cent.

Eosinophiles: none.

Macrolymphocytes or primitive cells· 80 to 90 per cent.

(Non-granular mononuclear cells with voluminous protoplasm, basophilic, and with clear nuclei)

(*e*) Lymphosarcoma.—In this condition the lymphatic tumor, generally located in the neck, shows rapid progression, attains a considerable size within a few months, and produces a rounded prominence covered with a pronounced network of veins, sometimes ulcerated, and capable of leading to copious hemorrhage.

Blood Examination.—The blood shows little change. There is a moderate degree of leucocytosis with excess of polymorphonuclears, with or without eosinophilia.

(*f*) Lymphatic granulomatosis.—Enlarged glands form masses of varying size, running a rather rapid, malignant course.

Blood Examination.—Generally there is a leucocytosis of 30,000 to 50,000

Differential Count —Polymorphonuclear leucocytes increased

Eosinophilic polymorphonuclears increased

Myelocytes sometimes present

* * *

Before concluding this section a word or two must be said concerning the moot question of the lymphatic temperament or diathesis, now needlessly obscured by discussions as to terminology and pathogenesis (inter-relationship—as yet uncertain—of scrofula, the lymphatic diathesis, tuberculosis, and "arthritism").

Clinically there is no doubt of the existence of a lymphatic diathesis Children in general, and certain children in particular, exhibit a marked tendency to excessive reactions on the part of the lymphatic channels and nodes, and in some of them, this tendency is expressed in a variety of clinical manifestations, chiefly involving the skin and mucous membranes, which, in the aggregate, justify us in speaking of a lymphatic temperament—an actual diathesis, constitution, or special morbid predisposition—sometimes called *lymphatism.*

Nucleated red cells are present in varying numbers.

(*d*) Acute leukemia.—The various groups of lymph-nodes, particularly the cervical and submaxillary, are involved, but

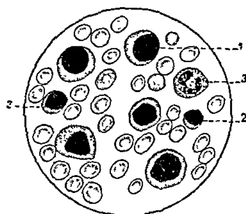



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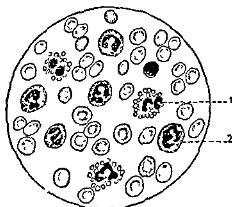



Fig 716—Lymphosarcoma *1* Eosinophile *2*. Polymorphonuclear neutrophile

Hemorrhages, pronounced anemia, and fever.

Blood Examination.—Cell Count.—The red cells show a marked decrease

# GLYCOSURIA. [γλυκύς, *sweet substance;* οὐρεῖν, *to urinate. Presence of sugar in the urine.*]

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Since uranalysis should be carried out as a routine in the process of examining a patient, glycosuria is a condition which ought not to be overlooked Even accidental or alimentary glycosuria is almost certain to be detected if the test for sugar is repeated at each examination, *as it should be*

In the event of doubt, glycosuria may be clinically considered to exist whenever there is distinct reduction of Fehling's solution (see *Technical procedures*)

The following figures will give an idea of the frequency with which glycosuria clinically occurs:

Out of 2000 subjects of both sexes suffering from various chronic disorders examined in the author's office, 106, or approximately 5 per cent, showed glycosuria; in two-thirds of these patients the condition had previously been overlooked

Out of 1000 military subjects, suffering for the most part from acute disorders, examined in a hospital, 6, or approximately 0 5 per cent, showed glycosuria; the condition had previously been overlooked in 4, *i e.*, two-thirds of the cases.

* * *

**Diabetes mellitus** is a clinical symptom-complex characterized by *permanent,* or at least *lasting, glycosuria, usually associated with polyuria, polydipsia, polyphagia, and autophagia,* none of these symptoms being, however, necessarily present in all stages of the disease, while the essential, characteristic manifestation is *habitual glycosuria with hyperglycemia*

Diabetes is put down as a **clinical symptom-complex and not a disease,** because the diabetes or permanent glycosuria is not always due to the same cause nor attended with constant pathologic evidences—as is also true, indeed, of temporary glycosurias.

Clinical observation and experimental work show that *temporary or permanent (diabetic) glycosuria* may be produced either:

With exclusive reference to the actual clinical evidences, these manifestations may be grouped as shown in the subjoined table:

**Clinical Manifestations of the Lymphatic Constitution.**

| | PRIMARY | SECONDARY, WITH A TENDENCY TO RECURRENCE |
|---|---|---|
| **Skin** . . . . . | Scaly eruptions Intertrigo Prurigo | Eczema. Impetigo. Multiple abscesses. |
| **Mucous membranes** . . . | Desquamative processes and various evanescent catarrhal states. | Sore throat<br>Pharyngitis<br>(Gastro-enteritis)<br>Coryza (hay fever)<br>Laryngitis (false croup)<br>Bronchitis (asthma),<br>Conjunctivitis.<br>Blepharitis.<br>Balanitis<br>Vulvo-vaginitis |
| **Lymphatic structures** | Hypertrophied palatal and pharyngeal tonsils Enlarged cervical, inguinal, axillary, or other glands | |

on by the insidious intoxications by *illuminating gas* and *carbon monoxide.*

The foregoing simple etiologic classification gives a sufficient understanding of the various well-known theories as to the causation of glycosuria, *viz.*, the hepatic theory, the pancreatic theory, the nervous theory, and the glycolytic theory These theories hold good in certain individual cases and explain certain clinical forms of glycosuria, but are not sufficiently comprehensive They apply in some forms of the condition, but not in all.

As matters now stand, if one takes into account all known clinical and experimental data, it becomes necessary either to limit one's efforts to special studies of each of the innumerable varieties of glycosuria or, adopting broader conceptions of the pathogenesis, to put it down as a fact that the diabetic symptom-complex is the obvious organic expression of a lesion or disturbance of function at some point of the glycotrophic nutritive system

This glycotrophic nutritive system, which is highly complex, is governed and coordinated by the organic cerebrospinal nervous system, which insures functional cooperation in this system, thus rendering it possible to have a *glycosuria of nervous origin through glycotrophic incoordination.*

The glycotrophic system consists essentially of a group of glandular organs, *viz.*, the liver, pancreas, and in fact the entire digestive tract, the adrenals, the thyroid, etc, charged with the task of elaborating sugars, presiding over their conservation (glycogenesis) and destruction (glycolysis), and whose overactivity or insufficiency, inducing disturbance of sugar nutrition, brings on a *diabetes of glandular origin*

Yet this glycolytic property, while more especially possessed by certain individual organs, appears to be a functional attribute of cell nuclei in general, so that any general disturbance of cell nutrition—usually combined, indeed, with the glandular disturbances above mentioned—may bring on a form of glycosuria that may properly be described as a *dystrophic diabetes of arthritic or toxic origin*

Brief reference may here be made to the ingenious theory involving the rôle of the endocrine glands (thyroid, adrenals, and, in part, the pancreas). This theory presupposes that the glycolytic

1. By **hyperglycophagia** (so-called "alimentary glycosuria"), including *muscular hypoglycolysis* (due to insufficient exercise)

2. By **disease of one of various organs.**

(*a*) **The liver.**—*Hepatic diabetes*, occurring in two forms (Gilbert and Carnot): *Excessive hepatic activity* (hypertrophy, congestive states, etc.), and *insufficient hepatic activity* (cirrhosis, obliteration of the portal vein, etc.).

(*b*) **The pancreas** (pancreatic insufficiency).—*Pancreatic diabetes*, a well-known condition clinically, and one also experimentally reproduced (von Mehring and Minkowski).

(*c*) **The kidneys** (renal insufficiency).—*Renal diabetes*, experimentally demonstrated through the production of phloridzin glycosuria. It is often familial, hereditary, prognostically favorable, but very persistent It is characterized by normal glycemia with permanent glycosuria. There is lowering of the threshold of elimination of glucose

(*d*) **The adrenals** (hyperepinephria).—*Adrenal diabetes*, experimentally demonstrated through epinephrin glycosuria.

(*e*) **The thyroid** (hyperthyroidia).—*Thyroid diabetes* (hyperthyroidia, Graves's disease), the actual occurrence of which is now under discussion. Mere coincidence is held to account for a number of cases

3. By **lesions of the nervous system.**—This was demonstrated experimentally by Claude Bernard in 1849, sugar appearing in the urine upon production of a lesion of the medulla.

(*a*) Organic disease, especially bulbospinal: Brain tumors, general paralysis, disseminated sclerosis, tabes dorsalis, etc.

(*b*) Neuroses and psychoses: Chorea, Graves's disease, etc.

(*c*) Traumatic lesions.

4. By **disturbed general nutrition.**

(*a*) **Neuroarthritic glycosuria**, dependent upon a chronic disturbance of general nutrition, usually inherited and variously combined with the "arthritic" disorders, *viz.*, gout, obesity, lithiasis, etc.

(*b*) **Toxic states**, acute or chronic. Those clinically most familiar, though generally evanescent, are caused by *chloroform* or *chloral hydrate*; the most important, however, because frequently overlooked, are the more or less lasting forms of glycosuria brought

which he wrongly deemed of little consequence; he had taken up his abode directly at the factory, whereas previously he had walked to and from the factory twice daily, thus covering a distance of 8 kilometers. Upon finding out this fact, I recommended that he take a walk for two hours each day. I also adjusted his diet. The glycosuria disappeared"

Such easy cases as this are exceptional; yet they are sometimes met with in practice The above example shows how intelligent and painstaking an inquiry is required before prescribing treatment.

A clinical classification of cases of diabetes, however imperfect it may be with our present restricted knowledge, is of marked service in defining the general lines of antidiabetic treatment in the various cases.

For want of a better one, the following least defective, oldest, and most practical classification will here be adopted

**Stout Diabetics, without Impairment of Nutrition**—These are usually cases of "neuro-arthritic" diabetes, or hepatic diabetes through overactivity of the liver; this is the diabetes of plethoric or of florid gouty persons with enlarged and congested liver, a sluggish intestine, and taking but little exercise Such diabetics generally appear very well nourished, stout, red complexioned, and show marked endurance; they eat and drink heartily and are in a general sense high livers.

In these patients the body weight is definitely above the average, and the same is true of the daily output of urea, which exceeds 0 40 gram per kilogram of weight; the glycosuria is of intermediate degree, ranging from 0 to 60 grams. If the urine is systematically collected at fairly regular intervals after and between meals, the sugar elimination will be found to be intermittent, or, if it is constant, a marked recrudescence after meals will be noted.

This is the commonest type of case, and these are also the most favorable cases from the standpoint of treatment, proper and well planned general hygiene and diet being sufficient for successful results Marked benefit accrues from restriction of the total intake of food, special reduction of carbohydrates, and systematic exercise.

**Thin Diabetics, with Impaired Nutrition**—This is the diabetes of young subjects and of those with serious disease of the pancreas or certain lesions of the liver

reaction in the cell nuclei can occur only in the presence of catalyzing reagents or complements set free in the system by the endocrine glands; in the absence of these indispensable complementary ferments, the glycolytic reaction is held not to occur, glycosuria therefore resulting. In truth, this theory, while opposed by the actual known instances of diabetes induced by hyperepinephria and hyperthyroidia, is based upon the definite observations well described by Minkowski and leading to the conclusion that glycosuria may be brought on by suppression of the internal secretion of the pancreas, which in the normal animal proceeds to exert its action upon the other glycotrophic organs through the medium of the circulation

At all events, the brief causal and pathogenetic review just presented will have imparted some conceptions as to the occurrence of the *syndrome of diabetes,* accounted for by one of a variety of causes, and the causal treatment of which—the only rational means of therapy—should be adapted to each clinical form.

However limited our knowledge may as yet be in this connection, it is thus nevertheless indispensable to make a systematic study of each case and try to trace back the initial cause of the nutritional disorder.

"Before undertaking to treat a diabetic subject, one should study the case with care not only as regards the existing symptoms but also as regards the patient's habits and character. The results obtained depend, indeed, upon a host of circumstances apart from the nature of the diabetic disorder *per se* The patient's occupation and tastes and the general type of his associates frequently offer hindrance to successful results from the physician's advice."[1] (Le Gendre)

This careful preliminary investigation will sometimes enable the physician very readily to dispel incipient diabetes. "My advice was sought a few years ago," wrote Lépine, "by a manufacturer about fifty years of age, free of inherited morbid taints. He was leading a normal life and was not subject to worry; his diabetic condition had set in about two years before

"After prolonged questioning I finally learned that during the preceding three years he had made a change in his daily routine

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[1] Diabète in *"Traité de médecine,"* vol i, Masson et Cie.

complex in their pathogenesis and not precisely compatible with the groupings already referred to. In these cases clinical investigation should be especially painstaking and intelligent, since a rational and sometimes effectual causal treatment may be worked out upon discovery of the exciting cause of the condition.

The author is well aware that the above mentioned groupings are arbitrary and are not adequately supported by clinical or pathologic data; yet they are, for the present at least, worthy of adoption from the practical standpoint, *i e*, for prognostic and therapeutic purposes

The disease in these cases is attended with rapid loss of weight, progressive asthenia, and a tendency to cachexia. The general appearance is one of physical debility, and resistance to fatigue is slight.

The body weight is distinctly below the average; polyuria and glycosuria are very marked, the former amounting to 3 liters a day and the latter to over 100 grams. The amount of sugar excreted may exceed the intake of carbohydrates. Examination of the urine collected at regular intervals shows that glycosuria is constantly present and that the influence of the meals on it, though present, is much less marked than in the preceding group of cases

These are the worst cases from the standpoint of treatment. Often it is impossible to arrest denutrition and prevent cachexia. The prognosis is most unfavorable.

**Nervous Diabetes.**—This group includes, indeed, several very different forms of the disorder; but, if one excludes the cases manifestly dependent upon some obvious lesion of the nervous system and in which the glycosuria is to be considered only as a bulbar manifestation of the organic disorder present, there remains a rather clear-cut clinical type, *viz.*, the diabetes of city dwellers, business men and others overburdened with mental work or continually subjected to emotional impressions and occupational stress and worry The predominating clinical feature in these cases is nervousness and irritability The urinary evidences are likewise very changeable; the polyuria and glycosuria vary markedly from one week to another or from one day to the next, without apparent relationship to the diet, and an obvious, direct connection can be made out between the degree of overwork and worry and the glycosuria and general condition of the patient. Oxaluria and phosphaturia are frequently present in these cases.

Obviously, in these subjects, general hygiene, mental rest if possible, country life, a regular mode of living, and nervine medication are the chief therapeutic indications

The above varieties having been enumerated, there remain certain *indefinite cases*, of varying etiology and symptomatology, such as post-infectious diabetes, toxic diabetes, traumatic diabetes, etc.,

complex in their pathogenesis and not precisely compatible with the groupings already referred to. In these cases clinical investigation should be especially painstaking and intelligent, since a rational and sometimes effectual causal treatment may be worked out upon discovery of the exciting cause of the condition.

The author is well aware that the above mentioned groupings are arbitrary and are not adequately supported by clinical or pathologic data; yet they are, for the present at least, worthy of adoption from the practical standpoint, *i e*, for prognostic and therapeutic purposes.

# HEADACHE (CEPHALALGIA).

[κεφαλή, *head;* ἄλγος, *pain, headache.*]

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Headache is one of the commonest of clinical symptoms A definition of the term **cephalalgia** is scarcely necessary; in a general way, it refers to pain, generally diffuse in character, experienced in any portion of the cranial region.

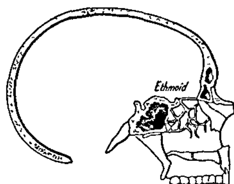



Fig 717—Cut showing the close anatomic relationship existing between the frontal, ethmoid, and sphenoid sinuses and the covering membranes of the brain. The ethmoid sinuses are diagrammatically represented.

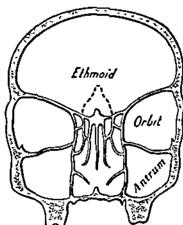



Fig. 718—Cut showing the close anatomic relationship existing between the ethmoid sinuses and the covering membranes of the brain

This region includes:

1. The *cranial contents.* Cerebrum, cerebellum, the brain membranes, and the intracranial vessels and nerves.

2. The *cranial cavity with its annexes:* Frontal, maxillary, and ethmoid sinuses, and the auricular and orbital cavities.

3. The *pericranial tissues* Pericranial muscles, insertions, and fasciæ (frontal, occipital, and temporal), and in particular the occipital (nuchal muscles) and temporal muscular masses; also the skin and cellular tissue of the frontal and temporal regions and of the scalp.

All these tissues, with the exception of the brain itself, are provided with sensory nerves; furthermore, while the brain itself is actually insensitive, the brain arteries are provided with sympathetic plexuses, and the dura mater and pia mater and their extensions are abundantly innervated; thus, many deep-seated brain disorders, *e g.*, tumors, may give rise to inveterate headache, possibly through meningeal or vascular irritation

This structural complexity accounts, at least in part, for the exceeding frequency of cephalalgic reactions, of which even the subjoined enumeration will give only an incomplete idea:

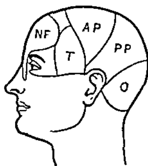



Fig 719—Head's cranial zones *NF*, nasofrontal. 6th upper thoracic segment Disorders of the pulmonary apexes and of the base of the heart. *T*, temporal 7th thoracic segment Disorders of the bases of the lungs, the left ventricle, and the upper gastric region *AP*, anterior parietal: 8th thoracic segment *PP*, posterior parietal. 9th thoracic segment *O*, occipital 10th thoracic segment

1 The *scalp itself* may be the source of the morbid impulses, as from parasites, wigs, hats, or dyes.

2. The *muscle mass* and fasciæ of the nuchal region may be the seat of fibrous deposition, of inflammation, or of painful infiltrations the importance of which will later appear

3 The *cranial sinuses*, frontal, ethmoid, and auricular (middle ears), by virtue of their contiguity to the nasopharyngeal mucous membrane, are particularly exposed to the catarrhal states and infections which so often affect the latter surface

4. The *intracranial membranes* (meningeal and vascular) are likewise frequently subject to congestive or inflammatory painful conditions.

5. The *trigeminal region*, rendered hypersensitive, it seems, by the refinements of civilization, is subjected to the abnormal irritation of affections of the eyes, nose, and teeth It might further be stated, apparently with sufficient justification, that the chief center of cephalic sensation, "the headache center," if one may use such an *expression, is principally made up of the "trigeminal centers."*

6. Many clinical observations compel recognition of the occurrence of *reflex headaches* brought on through morbid excitation of *extracranial regions*, sometimes very remote, probably owing to more or less intimate connections between the pneumogastric centers and the fifth pair. Thus, Head has described certain cephalic zones which he considers particularly "exposed" and painful in the presence of disorders of the thoracic or abdominal viscera Diseases of these viscera are well-known to cause hyperesthesia and reflex pain in certain definite zones of the thoracic and abdominal walls. If this pain exceeds, however, a certain degree of intensity, a similar hyperesthesia and pain tend to set in in a corresponding *cranial zone The general law of pain distribution,* as conceived by Head, is as follows· The higher up the affected portion of the trunk, the more anterior the affected zone of the encephalon. Thus, according to Head, the relationships existing between the trunk and head may be given as follows:

Six upper thoracic segments, naso-frontal area
7th thoracic segment, temporal area (held to be one of the most frequent types).
8th thoracic segment, vertical (anteroparietal) area.
9th thoracic segment, parietal area
10th thoracic segment, occipital area.

The hyperesthetic zone of the seventh thoracic segment, which corresponds to a visceral disorder at the base of the lungs, the upper portion of the stomach or the left heart, including more particularly the mitral region, is, indeed, often attended with temporal headache.

Reflex headaches of more remote source, *e.g.*, from the uterus or ovaries, have also been reported.

7. Lastly, the close connections existing between the cortical and subcortical centers of head sensation and the special sense centers (visual, auditory, *gustatory, and olfactory in particular*), as well

as the general and special hyperesthesia of hypercivilized subjects, account for the headaches of special sense origin induced in algiç-hyperesthetic persons by marked or prolonged special sense excitation, *e g*, too much light (as illustrated in the headache and neuralgia met with on the Côte d' Azur, or Southern Coast of France), sharp, discordant sounds, strong or nauseous odors, unpleasant tastes, etc

If one considers, furthermore, that all of the foregoing headache-producing stimuli may be direct or indirect, circulatory, inflammatory, toxic, etc, a conception will be had of the very great frequency of the symptom headache, the multiplicity of its causes, and the practical impossibility of supplying a complete, practical, and logical classification

For want of a better classification and in full knowledge of the attendant shortcomings and of the frequently artificial nature of distinctions thus made between different groups of headaches, the following classification, which is the least inconvenient, will be adopted herein

*Headaches of toxic and toxinic* (infectious toxins) *origin.*

*Headaches of neuralgic and neuropathic origin*

*Headaches due to pressure* (inflammatory, meningoencephalitic, or neoplastic).

*Headaches of reflex origin*

*Headaches of muscular origin.*

**Headaches of toxic origin** are very common and often difficult to diagnosticate, being highly variable as to type, severity, localization, and duration

In this group may be mentioned the **prodromal headache of infectious diseases.** In general these are poorly localized and of intermediate severity, they usually diminish in the morning and show exacerbation in the afternoon and evening; they are often associated with a gradual rise in the temperature; after the prodromal period they generally subside or diminish This prodromal type of headache is particularly marked in *typhoid fever, malaria,* and *influenza.* The first of these disorders may be suspected upon careful examination of the temperature curve and search for the other prodromal signs (epistaxis, dizziness, diarrhea, appearance

of the tongue, enlarged spleen, etc.), and its existence may be later confirmed by the Widal test and blood culture. Malaria is recognized from the periodicity of its febrile paroxysms and their characteristic successive stages; examination of the blood for malarial parasites may definitely settle the question Influenza is generally recognized from the existence of an epidemic at the time, the sudden onset, the respiratory catarrh, and the rapid course of the disease.

**Headache of uremic origin** seems to be most pronounced in the presence of *azotemia.* Often a comparative remission in the mornings will be noted. Frequently attending the digestive form, it is accompanied by nausea, vertigo, mental confusion, and sometimes vomiting; it frequently presents the appearance of migraine; high blood-pressure is almost constantly present; determination of the blood urea will definitely settle the matter. *In the high pressure forms without marked azotemia,* which seem to occur rather frequently in interstitial cases with excessive output of urine of low specific gravity, without albuminuria, but with markedly high blood-pressure, the *toxemia* is often less pronounced, and the headache is perhaps less dependent upon this factor than upon the lack of elasticity of the arteries with increase of the blood-pressure in the cranial cavity. Such conditions induce headache which varies with every change in the circulation and frequently assumes a pulsating character Rénon has called attention to a special form of morning headache occurring in high pressure cases and accompanied by polyuria, excessive output of urine at night, acute albuminuria, and hypertrophy of the left ventricle.

The **headache of stercoremia or constipation** is generally of the toxic type, and the frequent association of the cause with the effect is usually known to the patient himself.

A much more severe variety is the **bilious headache** which, in some individuals, recurs at irregular intervals and ranges from a simple persistent feeling of frontal weight to a lancinating, throbbing, boring pain. It is commonly associated with nausea and vomiting; the vomitus first brought up consists of more or less digested food, and that appearing later, of mucus and bile. This type of headache is due partly to the presence of toxic substances in the blood, but mainly to the brain congestion resulting

from repeated vomiting. Very common and annoying concomitant symptoms are palpitations and vertigo.

A form of headache recurring regularly in the morning, generally of slight intensity, and passing off after breakfast and a walk in the open air, is that due to *insufficient ventilation of the bedroom.* In such cases the gas pipes and chimneys should be examined with great care since, aside from the fatal risks which an intoxication of this sort may entail (as exemplified in the death of Zola and of Tarbé des Sablons), a mild but continuous intoxication may in the course of time induce a very obstinate type of headache together with marked alterations of the blood. Apparently also belonging in this category is the *inveterate winter headache of city dwellers,* coextensive with the cold season of the year and artificial heating of the houses, and absent throughout the warmer period and during life in the country

*Toxic headache due to tobacco or alcohol* is, as a rule, easily recognized.

Lastly, mention may be made of the *headache, sometimes very characteristic, of cases of low blood-pressure* or *hyposphyxia* (see *Low blood-pressure*)—an *occipital variety of headache which is enhanced by recumbency,* obstinate, resistant to all the usual treatments for headache, not influenced by seasonal or digestive factors, and always associated with low pressure and a relatively high blood viscosity

The term **headache of nervous origin** is manifestly only a makeshift used to designate headaches the cause of which appears to reside in a functional disturbance of the nervous system *per se,* although as a matter of fact careful clinical analysis nearly always leads to the detection of some proximate cause, reflex, congestive, anemic, or toxic

The most characteristic form is **migraine**, a unilateral type of headache recurring at regular or irregular intervals in plainly neurotic patients whose family history reveals the frequency of migraine among their forebears and collateral relatives, especially women Migraine is a very severe type of headache of the boring, throbbing type, generally accompanied by very distinct ocular symptoms. Sometimes the pain begins in one eye and

extends over the whole of the same side of the head; at other times, the converse condition occurs. The patient often experiences an impression as of luminous particles in the affected eye; the skin vessels are engorged, and there is commonly nausea and vomiting.

These attacks may last several days and are regarded by neurologists as representing "nervous discharges" for the production of which an inherited predisposition as well as an exciting cause are required. In very many cases there are present refractive errors and disturbances of ocular motility which demand careful examination, since much may be done in this direction to reduce the frequency of the attacks. While migraine may, like epilepsy, be looked upon as the result of an inherited nervous instability, it is none the less worthy of note that, in a given subject, the attack is often brought on by the same cause. The treatment is very difficult, and it is a source of great consolation for the patient to know that his attacks will become much less frequent during the second half of his life

Recognition must be given to the occurrence of a **headache of the migraine type of pituitary origin,** detection of which is facilitated by observation of the *ordinary signs of disturbed pituitary function, viz.,* increasing coarseness of the features, thick lips, prognathism, thick eyebrows, mustache in women, hairiness of the body and extremities, tendency to acromegaly, high blood-pressure, etc

Administration of preparations of the whole pituitary gland generally causes this type of headache and the other attendant symptoms to disappear.

**Facial neuralgia** is often attended with pain which may suggest cephalalgia The pain may be severe, lancinating, of sudden onset, and be accompanied by tender points along the course of the affected nerve. In bad cases, pronounced local edema may be superadded One should always bear in mind the fact that the neuralgias and, in a general way, all the headaches of nervous origin may be purely toxic or anemic.

**Neurasthenia and hysteria** are often attended with headache, the distinguishing feature of which is a feeling of pressure or numbness at the vertex, or of compression or constriction over

the lateral regions ("en casque"). One may here recall, moreover, the variety of neurasthenia so well described by Krishaber under the name *cerebrocardiac neuropathy*, owing to the predominance of cerebral (headache, insomnia, and depression) and cardiac (palpitations, tachycardia, angor, etc.) manifestations.

Usually there are found in combination with the headache neuropathic symptoms such as exhaustion on slight effort, alternating periods of exaltation and depression and, in a general way, nervous irritability. One should not forget, as already pointed out, that these headaches of so-called "nervous" origin and their proximate causes themselves are often the expression of some form of reflex irritation of a weakened nervous system, and that both headache and neurasthenia may actually be the result of a latent hyperesthesia of the eyes, nasal mucosæ, or stomach.

**Pressure headaches** may be the result of
**Inflammation.** **Meningo-encephalitis, syphilis.**
**Tumor.**
**Endocranial abscess.**
**Glaucoma.**

The differential diagnosis in these forms is often a matter of great difficulty and sometimes requires the assistance of a neurologist As a rule, they are characterized by their constancy, increasing severity, and nocturnal exacerbations; they are accompanied by fever in the acute diseases, such as **meningitis**; exceptionally, however, even in the case of **brain abscess**, they may be attended with subnormal body temperature Examination of the cerebrospinal fluid and testing for Kernig's sign are indicated in all instances. Marked assistance may be had, both as regards causal diagnosis and localization of the disease focus, from examination of the fundus of the eye or observation of a localized paralysis.

A very severe form of headache, often mistaken for neuralgia, is that due to acute glaucoma. The possibility of this diagnostic error is enhanced from the fact that there is frequently edema with points of hyperesthesia around the orbit and the patient thinks he sees luminous particles. Such a mistake is attended with serious consequences, for glaucoma, even when

properly treated, may lead to deep-seated visual disturbances, and an overlooked and consequently untreated glaucoma may induce complete blindness in a few hours. In glaucoma the eye is red and lachrymal secretion profuse, but the cardinal factors in the diagnosis are painful tension of the eyeball, a turbid and insensitive condition of the cornea, and wide dilatation of the pupil, which reacts poorly to light and to myotics.

Special reference should be made to **syphilitic headache,** which is of great diagnostic importance In this connection the two commonest types of this kind of headache should be recalled: 1. SECONDARY SYPHILITIC HEADACHE, deep-seated, with a feeling of weight, continuous, with vesperal exacerbations, apparently dependent, on the whole, upon an actual process of secondary syphilitic congestive meningo-encephalitis with cerebrospinal hypertension, as shown by lumbar puncture. 2. TERTIARY SYPHILITIC HEADACHE, circumscribed, boring, persistent, with nocturnal exacerbations, and dependent upon a gumma The Wassermann reaction and the efficacy of antisyphilitic treatment bring convincing proof to the diagnosis.

The headache of **brain tumor** is often localized, increased by percussion, and sufficiently severe to cause the patient to cry out; it appears in paroxysmal attacks and is generally accompanied by vomiting, dizziness, and pupillary disturbances.

**Headache of reflex origin.**—This form, as already implied, is steadily increasing in frequency, apparently because of the gradual heightening of civilization which, by increasing sensory acuity and developing specialized functions, is bringing about the formation of a series of normal or morbid reflexes which are absent among savages. *Prolonged, patient study of his case will enable the physician to find out whether a headache is reflexly* dependent upon some ocular, digestive, genital, or other source of irritation.

This type of headache is among the most frequent symptoms of **ocular disorders,** and such a cause may be suspected where the pain is particularly localized in the superciliary, frontal, or temporal region, and where, being absent on rising in the morning, it thereafter gradually increases with increasing use of the

eyes and becomes very marked upon constant use of the eyes for some task requiring minute ocular adjustment. The severity of such headache bears no relationship to the extent of the ocular disorder; slight refractive defects often cause much more severe headaches than marked defects. Patients frequently have obvious refractive errors without experiencing any symptom of eye-strain, owing to an instinctive compensatory reaction Thus, many persons with hypermetropia are able to see objects distinctly, without apparent ocular fatigue, by continuous contraction of the ciliary muscles If, however, they overwork or lose the hypertrophy of their ciliary muscles either by reason of abuse or through disease, vision becomes difficult and close work impossible.

Myopic persons, whose distant vision is improved when the ciliary muscles are completely relaxed and consequently almost atrophied, similarly suffer from frequent headaches whenever they undertake any continuous work requiring some accommodative effort. The same result is often brought about by overcorrection of myopia, when the latter is estimated solely with the objective procedures employed by opticians.

Very distressing headaches are also induced by even a slight degree of *astigmatism*.

Under normal conditions the ocular muscles maintain the eyeball in such a position that the rays of light from a distant object fall directly upon the macula lutea without requiring any exertion on the part of the individual. Frequently one group of muscles is too strong or too weak, so that a parallel position of the two eyeballs is obtained only by dint of overcontraction of the weaker muscles Generally the muscle is able to carry out its task and the tendency to deviation can be detected only by special tests; the constant exertion entailed by binocular vision may, however, lead to a number of distressing nervous symptoms, particularly headache Sometimes the weaker muscles become momentarily insufficient and temporary strabismus is then noticed; in other instances, the strabismus is permanent Where there is continuous, permanent strabismus, the patient gets in the habit of disregarding completely the image from one of his eyes, and great patience is then required to convince him

that he "sees double"; he is not making any effort to combine the two images and hence does not suffer from asthenopia and generally has no headache.

Headache of nasal origin is less common than that due to eye-strain. It is generally localized in the frontal region and associated with some obvious nasal disorder. It is the result of irritation of the terminals of the fifth pair in the nasal mucous membrane. For example, swelling of the mucous covering of the turbinates in *acute rhinitis* is accompanied by a dull headache, particularly if the nasal cavities are too small to permit of this mucous swelling without morbid pressure; this sort of headache is very often relieved by the use of vasoconstrictor astringents, such as cocaine or adrenalin Headache recurring regularly at certain seasons or when the wind blows from a certain direction is often of reflex nasal origin. In *subacute rhinitis* the headache is more marked in the morning owing to the accumulation of mucous secretions in the nasal cavities during the night. An *ulceration of the nasal mucosa* exposing the nerve terminals may be the cause of a reflex headache. This is also, as is well known, an important symptom in *sinus inflammations*.

Head, as already mentioned, made a special study of the zones of cutaneous hyperesthesia and the corresponding cranial headache zones as they occur in disorders of the thoracic and abdominal viscera governed by the vagus nerve. The following table epitomizes his conclusions:

**Head's Zones.**

| Zones of Cutaneous Hyperesthesia | Corresponding Cranial Zones. | Deep Viscera Associated with These Zones |
|---|---|---|
| 3d and 4th cervical | Naso-frontal. | Apex of lung, liver, stomach, aortic orifice. |
| 2d, 3d, and 4th dorsal | Pretemporal. | Lung, heart, aortic arch |
| 5th and 6th dorsal. | Fronto-temporal. | Intermediate portion and base of lung, left ventricle, upper gastric region. |
| 8th and 9th dorsal. | Antero-parietal and parietal | Stomach, liver, upper portion of small intestine. |
| 10th dorsal. | Occipital. | Liver, intestine, ovaries, testicles |

Utero-ovarian disorders are a frequent cause of reflex headache, whether the condition present be ulceration or displacement. This type of headache is often localized in the occipital region and becomes accentuated during the menstrual periods. Indeed, headache is so frequently present at these periods that it seems almost a normal accompaniment of the menstrual process

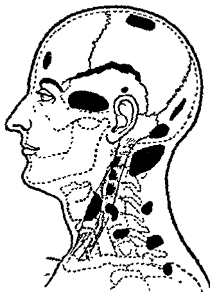


Fig 720—Elective areas of fibrous thickening over of the skull, nucha, and neck.

Lastly, many persons are subject from time to time to slight headaches, perhaps partly reflex, as a result of *exposure to cold* or of an *emotional impression* or *dietary indiscretion*

The foregoing list, in spite of its already tiresome length, is far from exhausting the possible causes of headache. There is one group, not yet referred to, which has been the subject of numerous investigations in late years, *viz*, that of the **headaches of muscular origin**, seemingly dependent upon *connective tissue infiltration of the muscles of the neck, particularly of the nuchal region, and of the head*, nearly always accompanied by excessive muscular tone with a tendency to rigidity and frequently an arthritis

sicca of the vertebral articulations. Palpation of the back of the neck and particularly of the skull over the fascial insertions enables one directly to observe the presence of such fibrous thickenings. The headache in these cases may assume one of three types (Hartenberg):

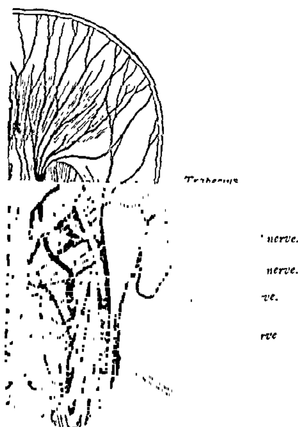



Fig 721 —Posterior branches of the cervical nerves (*Soulié*).

1. Frank migraine with unilateral pain, arterial throbbing, scotomas, and vomiting

2. Neuralgia, definitely localized and associated with the presence of tender points.

3. Indefinite headache with a feeling of weight or a crushing or dragging sensation.

Hartenberg, who has made a special study of this variety of headache, ascribes these infiltrations to a cervical myocellulitis. "I feel justified in incriminating," he states, "as the chief exciting cause

of these infiltrations, an insufficiency of arterial, venous, and lymphatic circulation, due, in turn, to insufficient muscular activity. Indeed, local exposure to cold and reduction of temperature, which reduce both tissue metabolism and circulation, distinctly favor the production of these lesions Again, I have observed them particularly in subjects leading a sedentary life, and those who take physical exercise are free of them

"Cellulitis thus appears to us as a species of tissue rust due to insufficient physical activity It constitutes a stigma of premature senility in overcivilized subjects who have replaced muscular labor by brain work."[1]

The series of causes above enumerated shows how difficult the differential diagnosis sometimes is In truth, often it is obvious and does not require any very prolonged examination, *e g.*, in the headache of infectious diseases, of acute meningitis, of migraine, etc In other instances, again, a painstaking clinical study is required On the whole, there are a certain number of points which should never be forgotten during such studies, and which, correctly illuminated, will yield a positive diagnosis in 95 per cent. of cases of obstinate chronic headache:

**History of the Illness.**

1 Are there paroxysms, sometimes at regular intervals (monthly), or with ocular disturbances, nausea, etc (migraine)?

2. Is the headache periodic, and accompanied by attacks of fever (malaria)?

3. Are there clear-cut past evidences of specific infection (syphilis)?

4. Have there ever been manifestations of a psychoneurosis (neurasthenia)?

Etc.

**During the clinical examination, one should always examine:**

1 The *eyes,* including the retina (albuminuric retinitis, vascular disturbances, evidences of intracranial disease)

The pupils (reaction to light, Argyll-Robertson pupil, showing a specific meningo-encephalitis).

The intraocular tension (glaucoma).

[1] HARTENBERG *Presse méd*, Feb 14, 1912, p 134

## HEADACHE.

| Causes. | History. | Duration. | Regular Recurrence of Attacks. | Fever and Signs of Infection | Blood-Pressure | Urine | Blood | Cerebro-spinal Fluid | Eyes | Nose. | Ears | Nuchal Muscles |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Infectious diseases. | ? | That of the fever. | Regular in malaria | + | — | Frequently albumin. | Leucocytosis | 0 | | | | |
| Uremia. | Nephritis. Albuminuria. | Weeks or months. | 0 | 0 | + | Albumin + | Blood urea + | Hyperazotemia. | | | | |
| Stercoremia. | Constipation Intestinal atony or spasm. | Chronic. | 0 | 0 | | 0 | 0 | 0 | | | | |
| Bilious Headache. | Cholemia. | Chronic, with paroxysms. | 0 | 0 | | 0 | 0 | 0 | | | | |
| Anoxemia. | Bad ventilation. Sedentary life. | Months, during the winter season of house heating. | 0 | 0 | — | 0 | Anemia | 0 | | | | |
| Migraine. | Neurasthenic heredity. Previous attacks. | 12 to 24 hours. | + | 0 | | 0 | 0 | 0 | | | | |
| Facial neuralgia. | ? | Days, with paroxysms. | 0 | 0 | | 0 | 0 | 0 | | | | |
| Psychoneuroses (neurasthenia, etc). | Psychasthenia. Vasomotor disturbances, etc. | Chronic. | 0 | 0 | | 0 | 0 | 0 | | | | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Meningitis.** | ? | Weeks. | 0 | + | | Occasionally albumin. | Leucocytosis? | + High pressure Cephalalgic changes. | | | | Rigidity of the neck. Kernig's sign |
| ***Brain tumor.*** | ? | Weeks | 0 | + | | Occasionally albumin. | 0 | High pressure | | | | |
| **Syphilis.** | Syphilis. | *Days or weeks.* | *Sometimes* nocturnal recrudescences | + or 0 | | Occasionally albumin | Wassermann + | + or 0 | | | | |
| **Ocular disturbance.** | Glasses worn Myopia, presbyopia | Chronic. | 0 | 0 | | 0 | 0 | 0 | + | | | |
| ***Nasal* disturbances (sinusitis).** | *Nasopharyngitis.* Polypi, etc | *Chronic,* with paroxysms | 0 | + or 0 | | 0 | 0 | 0 | | + | | |
| **Aural disturbances.** | Otitis | Chronic, with *paroxysms* | 0 | + or 0 | | 0 | 0 | 0 | | | + | |
| **Visceral disorders (reflex headaches).** | Pleuro-pulmonary. Digestive Utero-ovarian | Very variable | 0 | 0 | | 0 | 0 | 0 | | | | |
| **Indurative muscular headache.** | Sedentary life Insufficient exercise and fresh air. | Chronic | 0 | 0 | | 0 | 0 | 0 | | | | Areas of induration. Rigidity. |

2. The *ears,* middle and internal (otitis, brain abscess).

3. The *nose* and its *annexes* (maxillary and frontal sinuses).

4. The *temperature* (toxic-infectious headache).

5. The *blood-pressure* (headache of high-pressure cases, of uremia, of hyposphyxia)

6. The *urine* (albuminuric, indicanuric, or acetonemic headache).

7. The *blood* (determination of the blood urea for uremia, Wassermann reaction for syphilis).

8. If need be, the *cerebrospinal fluid* (high pressure and the white cell formula, *e.g.,* lymphocytosis in tuberculous meningitis and polynucleosis in ordinary meningitis, etc.).

9. The *muscle insertions* on the cranium and nuchal region (headache of muscular origin).

# HEMATEMESIS.

[αἷμα, *blood*; ἐμεῖν, *to vomit*. *Vomiting of blood.*]

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Hematemesis consists of the vomiting of blood. The presence of blood in vomited material is frequently obvious; if it is doubtful, blood should be tested for by the usual procedures employed for the purpose [See *Blood examination*. Microscopic examination (red cells, hematin crystals); spectroscopic examination (hemoglobin spectrum), chemical examination (Meyer's test)].

The diagnostic problem is put before the practitioner in the following terms:

A. **Is hematemesis taking place?**

I. **And first of all, is it blood that has been brought up?**

(*a*) If the vomited material is bright red blood, there is no difficulty in recognizing it, and all that is necessary is to exclude the possibility of hysterical simulation

(*b*) If it is **coffee ground** vomit, it may be confused (in rare instances) with the black vomitus of a patient who has taken in succession a preparation of ergotin and gallic acid and one of ferric chloride (ink formed in the stomach); also with *biliary black vomit.*

1. *Microscopic examination* shows more or less distorted red corpuscles and crystals of hematoidin or hematin.

2. *Spectroscopic examination* permits of easy differentiation

3 Frequently the *coexistence of intestinal hemorrhage* obviates the need of these procedures.

4 Sometimes there are only traces of blood.

In the latter event, the most serviceable procedure for clinical purposes consists in mixing in a test-tube some tincture of guaiac, ozonized oil of turpentine, and gastric juice If blood is present, even in small amount, a characteristic blue color will appear (see *Blood examination* Weber's test).

II. **It is blood. Where is the seat of the hemorrhage?**

(*a*) It is from the pharynx, the nose, or the mouth; inspection of these cavities will settle the matter.

(*b*) It is from the esophagus:

Former indications of disease of this canal (dysphagia, post-sternal pain, fluoroscopy after ingestion of bismuth) alone permit of establishing such a diagnosis.

The above causes having been excluded the following question, which is that of greatest clinical importance, arises:

(*c*) Did the blood issue from the stomach or the respiratory tract? Is there *hematemesis* or *hemoptysis?*

1. Particular import attaches to this question in view of the fact that entrance of a few drops of blood into the larynx in the course of hematemesis is sufficient to induce cough, and conversely, that in hemoptysis blood may be swallowed and then expelled in the act of vomiting. Accordingly, although the blood in hemoptysis is generally red, foamy, and mixed with air-laden mucous discharges, *this sign is absolutely unreliable.*

2. Consequently, the diagnosis is based on the *concomitant signs.* In hemoptysis the physician will note the customary evidences of disorders attended with this condition (tuberculosis, hyperemia, or apoplexy of the lung, or valvular heart disease); in hematemesis, gastric manifestations predominate (dyspepsia, epigastric pain, dilatation of the stomach, etc.)

3. The practitioner may be greatly puzzled in the event of simultaneous presence of gastric and pulmonary disease (*gastric ulcer and pulmonary tuberculosis*).

4. In theory, the differential signs of hematemesis and hemoptysis may be summarized thus·

| HEMOPTYSIS | HEMATEMESIS |
|---|---|
| History of lung disturbance. | History of a gastric, hepatic, or splenic disorder. |
| The blood is expectorated | The blood is vomited up. |
| *It is red, foamy, and frothy* | It is black, compact, and free of air admixture |
| It may be mixed with sputum | It may be mixed with bile or food débris. |
| The onset of hemoptysis is often heralded by a pricking sensation in the larynx | The onset of hematemesis is often heralded by a feeling of dizziness or faintness |
| It may be accompanied by nausea and pain in the chest | It may be accompanied by nausea and pain in the epigastrium |
| It is rarely followed by discharge of blood from the bowel | It may be followed by discharge of blood from the bowel |

## Ulcer and Cancer of the Stomach.

### Catheterization of the Fasting Stomach.

| | | |
|---|---|---|
| (a) Food stasis . .. . . .. | | Pyloric stenosis and, if true stasis, cancer of the pylorus |
| (b) No food stasis. | Wash water contains free HCl | Reichmann (probably pyloric ulcer). |
| | Wash water, with 1 per cent acetic solution, contains chemically demonstrable blood | Ulcer of the body of the stomach (simple or cancerous). |

### Examination of the Stomach after a Test Meal.

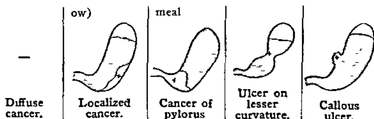
| | | |
|---|---|---|
| (a) Free HCl in excess | Ether capsule dissolved in less than one hour | Probable ulcer. |
| (b) Free HCl reduced toward 0 | Ether capsule not dissolved | Probable cancer. |

### Examination of Feces after a Milk and Vegetarian Diet.

| | | |
|---|---|---|
| Presence of blood chemically demonstrable | Blood disappears after a few days' rest | Probable ulcer. |
| | Blood still present. | Probable cancer |
| | Blood is present in feces but not in acidulated wash water from stomach | Duodenal ulcer or ulcer on duodenal aspect of pylorus. |

### Fluoroscopic Examination (Principal types)

| Small, contracted stomach | Filling-defect in stomach | "Amputation" of the py- | Stomach presenting a | Diverticular aspect (ap- |
|---|---|---|---|---|




| Diffuse cancer. | Localized cancer. | Cancer of pylorus | Ulcer on lesser curvature. | Callous ulcer. |
|---|---|---|---|---|

### Blood Examination.

Increased antitryptic power of the serum . . . . . . . Cancer.

### Cytologic Examination.

Microscopic examination of the wash water after gastric lavage
Study of centrifugation sediment.
Presence of neoplastic cells .. . . . ...... .. . .... Cancer.

**B. Hematemesis has occurred. What is its cause?**

**I. The hematemesis occurs under certain special circumstances, independently of any local disorder** which might facilitate discovery of the causes of the bleeding.

(*a*) **It occurs in the course of an infectious disease:** Hemorrhage-producing disorders such as scurvy, *purpura, hemorrhagic* smallpox, infectious endocarditis, typhus fever, plague, pernicious malarial fevers, grave icterus, and yellow fever.

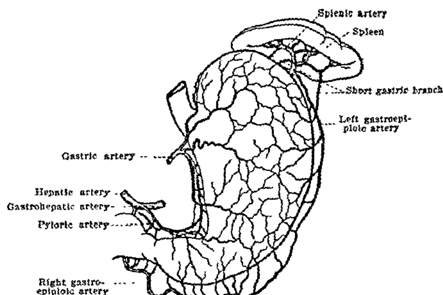



Fig 726—The arteries of the stomach.

(*b*) It may follow phosphorus or arsenic intoxication.

(*c*) It may be substituted for hemorrhoidal or menstrual bleeding.

(*d*) In predisposed subjects, especially *hysterical persons, it may come on following some pronounced emotional impression,* slight traumatism to the epigastrium, or exposure to cold. The diagnosis in these cases is based on:

1. Persistence of the disorder in spite of all treatment.
2. Relatively slight impairment of the general condition in spite of copious hematemesis.
3. The presence of permanent stigmata of hysteria, such as anesthesia, and contraction of the visual fields.

The fact should be borne in mind, however, that gastric ulcer is not uncommon in neuropathic subjects.

(*e*) Again, attention should be called to the possibility of hematemesis where persons are exposed to a *sudden reduction of atmospheric pressure*, as exemplified in the "Zenith" balloon disaster

(*f*) Lastly, mention may be made of the hematemesis of *uremia* and of unrecognized strangulated hernia in old persons (Robin).

II. In cases of exactly opposite type, the hematemesis is recognized as being of local origin, and the seat of the hemorrhage is localized in the stomach, its annexa, and other organs.

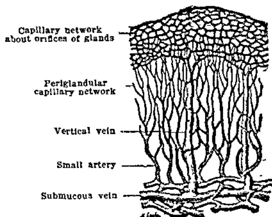



Fig. 727—Blood-vessels of the gastric mucous membrane (*Brinton*).

(*a*) Stomach: 1 May be mentioned as *occasional causes* Internal or external traumatic injuries, miliary aneurysms, thrombosis, embolism, amyloid infiltration, and venous stasis of cardiac origin

2. As *common causes* Cancer and ulcer (see p 995)

3 Some difficulty attends the differentiation of hematemesis due to ulcerations of the stomach in the presence of chronic gastritis, particularly of uremic origin All the symptoms combine to mislead the clinician; under these circumstances Robin's precept should be called to mind "You should make a diagnosis of cancer only when you cannot do otherwise"

(*b*) Auxiliary digestive organs.

1. Diseases of the liver.

*In cirrhosis of the liver,* especially atrophic cirrhosis, hematemesis sets in rather early, in the pre-ascitic period, and is then associated with the usual clinical signs of the pre-cirrhotic stage; sometimes it is very copious, and in some instances it has been known to cause death. It seems to be dependent both upon varicose vessels of the esophagus and upon altered nutrition Undoubtedly it occurs very often. Like the succeeding forms, it is largely secondary to the syndrome of high portal pressure (see Fig. 729).

*Hypertrophic cirrhosis* and *cancer of the liver;* mention should

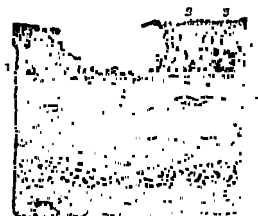


Fig 728—Section of gastric ulcer (*Dieulafoy*). *U*, ulcer formed at the expense of the mucosa *M* and the muscularis mucosæ *mm*, *a*, a submucous arteriole destroyed at the point *h*, where a large number of red cells are collected, the fatal hemorrhage has taken place at this point, *vth*, a thrombosed vein, *sm*, submucous layer, *mtr* and *ml*, muscular layer; *s*, serous coat; *1, 2, 3*, miliary abscesses in the depths of the mucous layer

also be made of *grave icterus,* which is attended with hematemesis for various reasons, some local and others general.

2. Pressure on the portal vein by tumors of the hilum or of neighboring structures (pancreas, etc ).

3. Embolism of the mesenteric, hepatic, or splenic arteries, *e g.*, in the course of infectious endocarditis.

(*c*) **Disorders of neighboring organs.**

1. Duodenum: See *Dyspepsia*

*Ulcer* of the duodenum may be attended with hemorrhage, in which the blood travels back toward the stomach. The diagnosis

is based on the frequency of intestinal hemorrhage, the different seat of pain, and the result of clinical examination according to Meunier's procedure (see p. 75).

Varicose conditions of the duodenum occur under the same circumstances as varicose conditions of the esophagus or stomach (syn-

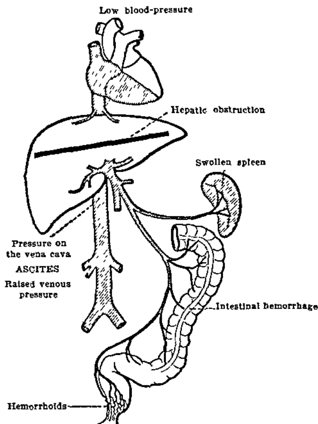



Fig 729—The syndrome of high portal pressure (portal hypertension).

drome of portal hypertension). *Ulcers* may also follow extensive burns of the body surface.

2. Esophagus:

*Varicose vessels* of the esophagus are met with particularly in cirrhosis of the liver or cancer. The customary evidences of esophageal tumor are present under these circumstances, *viz.*, dysphagia, pain, regurgitation of food, results of examination with the sound, etc.

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Fig. 728—Section of gastric ulcer (*Dieulafoy*). *U,* ulcer formed at the expense of the mucosa *M* and the muscularis mucosæ *mm*, *a*, a submucous arteriole destroyed at the point *h*, where a large number of red cells are collected, the fatal hemorrhage has taken place at this point, *vth*, a thrombosed vein; *sm*, submucous layer, *mtr* and *ml*, muscular layer, *s*, serous coat; *1, 2, 3,* miliary abscesses in the depths of the mucous layer

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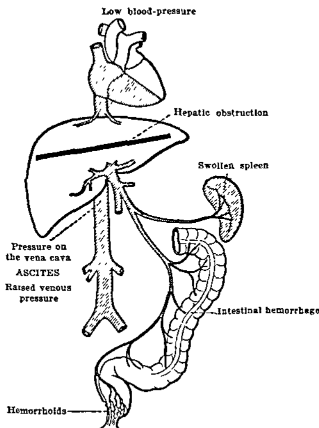



Fig 729.—The syndrome of high portal pressure (portal hypertension).

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2 ESOPHAGUS:

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| KIND OF VOMITING | CLINICAL HISTORY | CLINICAL SIGNS. | REMARKS. |
|---|---|---|---|
| **Ulcerative Conditions.** | | | |
| **Gastric ulcer.** Often copious vomiting of bright red blood. | Patient relatively young<br>History of dyspepsia with hyperchlorhydria<br>Characteristic late attacks of gastric pain | Appetite retained; delayed pain; hyperchlorhydria, more or less rapid restoration to health<br>Fluoroscopic examination (see p 995) | The black or **red color** of the vomitus in cancer or ulcer is by no means a certain indication; the color depends, as is well known, upon the length of time the blood remains in the stomach When due to the bleeding of a vessel ruptured by ulceration, the large quantity of blood extravasated is rapidly evacuated; the blood vomited up is red; this is frequently the case in ulcer<br>When due to the oozing from a tumor, the slow, continuous hemorrhage is evacuated only after some delay, and the blood vomited up is black, this is frequently the case in cancer. |
| **Gastric cancer.** Frequently vomiting of dark colored blood (coffee-ground). | Patient relatively old<br>History of ordinary dyspepsia or of ulcer<br>No attacks of pain for a long time. | Anorexia; loss of weight, vomiting, hypochlorhydria<br>*Tumor involving the stomach*<br>Fluoroscopic examination (see p 995). | |
| **Portal Hypertension.** | | | |
| **Cirrhosis of the liver** (varicosities of the stomach; erosions).<br>Frequently vomiting of dark colored blood, though sometimes red blood, in large amount. | History of a cause of cirrhosis (alcoholism, malaria, syphilis, etc.). | Liver small (atrophic cirrhosis) or large (hypertrophic).<br>Indications of long standing portal hypertension<br>Hemorrhoids; collateral circulation | These 2 forms of hematemesis present nearly identical features<br>They are in many respects alike as regards pathogenesis.<br>1. Portal hypertension; varicose veins of the stomach (gastric hemorrhoids). 2. Erosions or congestive ulcerations |
| **Alcoholic gastritis** (varicosities of the stomach; erosions).<br>Same features as regards vomited blood. | Alcoholism<br>Mucous discharges in the morning<br>Signs of chronic gastritis | Mucous discharges in the morning.<br>Tremor of the extremities.<br>Congestion of the liver. | The amount of blood vomited is variable There may be merely an oozing (dark colored hematemesis), or actual hemorrhage (red hematemesis). |

| | | | |
|---|---|---|---|
| Note.—Uremia causes the same disturbances (chronic gastritis, hematemesis, etc) Uranalysis and determination of the blood urea *will settle the question.* | | | All violent and protracted retching (hysteria, catarrhal states of alcoholics and smokers) may in the course of time cause minor attacks of hematemesis which are easy to *differentiate* |
| Splenomegaly. Same features. | | Enlarged spleen Anemia *Leukemia* | |
| *Heart failure.* Same features. | History of heart disturbance. | Cardiac and peripheral signs of heart failure *Passive congestion* of the liver | |
| Infectious or toxic diseases promoting hemorrhage (dyscrasic states) No special features | Those of the primary disease | Grave icterus, infectious endocarditis, scurvy, purpura, *hemorrhagic* small-pox, plague, yellow fever Phosphorus or arsenic poisoning. Uremia of the digestive, ulcerative type | It should not be overlooked that the *clinical classification* used in this table is in some respects arbitrary and defective Under many circumstances—*e g , in uremia and in* infectious diseases—hematemesis may be of threefold causation: 1. *Dyscrasic 2 Ulcerative. 3 Me*chanical (portal hypertension). |
| Trauma of the stomach. No *special features* | History of recent traumatism, usually a blow *in the epigastrium,* exceptionally a foreign body. | | |

Gastric ulcer, gastric cancer, and the syndrome of portal hypertension due to hepatic obstruction (as by cirrhosis, hyperemia, etc) represent, for practical purposes, *95 per cent of the causes of true hematemesis*

# HEMATURIA.

[ αἷμα—οὐρεῖν
*to urinate blood.* ]

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**Hematuria** is one of the commonest and most important manifestations of disease of the urinary tract For clinical purposes the term *hematuria* will be held to refer to cases in which blood in the urine is visible to the naked eye.[1]

The physician should, in the first place, make certain that hematuria actually exists by himself directly examining, if possible, the suspected specimen of urine obtained from the patient.

He should exclude, then, either by direct inspection of the urine or by microscopic or spectroscopic examination, *pseudo-hematuria due to ingestion of drugs* (see below) and *hemoglobinuria*, and should, furthermore, guard against making a serious error where blood from the female genital tract is mixed with the urine, as in menstruation or metrorrhagia.

The actual existence of hematuria having been positively established, he should next endeavor to ascertain the source of the hemorrhage and its cause, without waiting for further symptoms to appear

Hematuria often occurs abruptly, during apparent good health, in a subject previously free of any symptoms referable to the urinary tract. Under these circumstances it constitutes an alarm signal, practically a useful manifestation of a disorder as yet latent or circumscribed, early recognition and treatment of which may result in complete cure—which is all the more advantageous in that the more distressing phases of the disease will thus have been forestalled

---

[1] It should be borne in mind, however, that there may occur a microscopic hematuria, which sometimes plays an important part in the diagnosis of renal calculus, according as microscopic examination of the centrifugated urine, before or after walking, shows the presence of red blood cells in greater numbers in the urine collected after exertion or fatigue.

Too often, however, it happens that the physician, reassured by the absence of other clinical signs, rests content with the mere observation of the presence of hematuria; only much later, when the hematuria recurs, when it alarms the patient, and when other symptoms appear such as a local swelling, fever, and pain, does he think of carrying out a complete examination of the patient.

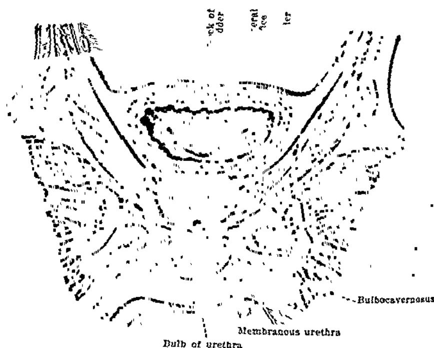



Fig 730

This mode of procedure is all the more to be deplored in that, thanks to systematic clinical study and especially to the modern means of examination, one is able very early, and *even in the absence of any other symptom*, to detect the cause of the bleeding.

In the presence of hematuria two questions arise· Where does the blood come from? and What is the cause of the hemorrhage?

It will be well to consider the special features presented by the hemorrhage according as it comes from one or another portion of the urinary tract, as well as the attributes afforded to it by its various possible causes

**I. The blood comes from the urethra or the prostate.**—The blood appears at the start of micturition. It is an initial hemorrhage, the blood in the urethral canal, washed down by the urine, appearing with the first drops of urine that pass out.



Fig 731—Sources of the internal pudic vein, *H*, and the vesical vein, *V* (*Farabeuf*) The pin passing through the lower and outer surface of the bladder represents the dividing line between the two venous currents, pelviovesical, *V*, and perineopudic, *H*

This *initial* type of hematuria is of small amount, and follows either a traumatized state of the urethral canal (acute urethritis, injury during coitus, rupture of the urethra, or false passage during catheterization) or a lesion of the prostatic urethra (varicose

vessels of the prostatic region, cancer of the prostate). The diagnosis is based particularly on the history.

Where initial hemorrhage is more pronounced, blood appears at the meatus independently of micturition The condition is then an actual **urethrorrhagia**, the amount of which bears a relationship to the seriousness of its cause. Such urethrorrhagia is met with chiefly as a result of rupture of the urethra, either from external traumatism (fall or blow on the perineum) or internal traumatism (serious false passage).

Sometimes blood coming from the posterior urethra or prostate independently of micturition passes back into the bladder, and if the bleeding is of considerable degree, the entire amount of urine may be discolored by it; upon inserting a catheter and washing out the bladder, however, the urine in the latter will rapidly become clear.

II **The blood comes from the bladder.**—Vesical hematuria may be **terminal** or **total.** *Terminal* hematuria, characterized by the appearance of blood, generally in moderate amount, at the end of micturition, at the moment when the bladder is completing its evacuation by means of a few forcible contractions, is indicative of a lesion of the neck of the organ, as in gonorrheal cystitis

Disorders involving the *body* of the bladder produce more abundant hemorrhage, the blood may in these cases be completely mixed with the urine, which issues red from the beginning to the end of micturition and may be deeply colored.

The *vesical origin of total hematuria* may be established as follows:

When the blood is derived from the bladder, the depth of discoloration of the urine increases as the close of micturition is approached If the patient is caused to urinate into three glasses, the first two glasses show less discoloration than the third; lastly—an important indication—a bleeding bladder is hard to wash out

Thus, given a patient with hematuria. A catheter is inserted and, upon slow injection of fluid into the bladder several times, the wash water tends to come back clear at the beginning of its expulsion, but as the bladder is further emptied, the later wash water shows increasing discoloration, which is the more

marked according as the bladder is allowed to empty itself more completely. If, furthermore, one waits until the bladder is thoroughly empty, the bleeding at once recurs, sometimes in the form of an outflow of almost pure blood.

*Vesical hematuria is met with*

(*a*) In trauma of the bladder; here the circumstances under which the hematuria arose point definitely to the source of the bleeding.

(*b*) In cystitis—tuberculous, calculous, or neoplastic cystitis, or simple hemorrhagic cystitis

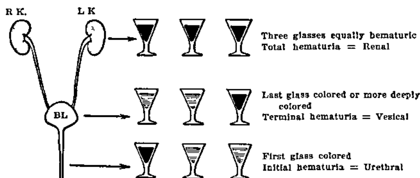



Fig 732

(*c*) In the absence of all signs of cystitis, in certain tumor formations—papilloma, angioma, and beginning cancer

(*d*) In some forms of congestive prostatic enlargement in which hemorrhage occurs into the bladder

**As a general rule,** the practitioner should not be satisfied with a diagnosis of hematuria of vesical origin based on the observation of other bladder signs such as pain, tenesmus, pyuria, etc.; bladder manifestations may occur in the presence of hematuria of renal origin, and to depend upon such evidences may lead to serious mistakes.

**The only absolute rule, which must be remembered as a definite axiom, is that any total hematuria believed to be of vesical origin points to the necessity of cystoscopic examination of the bladder.**

**III. The blood comes from the kidney or ureter.**—***Hematuria of renal origin*** **is a form of total hematuria** in which the urine

shows a uniform discoloration from the beginning to the end of the act of micturition. In contrast to that which occurs in vesical total hematuria, the wash water finally becomes quite clear or, if there is continuous oozing in the kidney, the results of irrigation will always be sufficiently marked to permit of immediate cystoscopy.

There are three great causes of renal hematuria which should at once come to the practitioner's mind, *viz.*, **tuberculosis, stone,** and **cancer.**

In each of these three disorders, bleeding may appear *abruptly*, without any previous or coexisting symptom. It is essential that the physician be familiar with this fact, since it is in these cases that early hematuria constitutes an actual benefit, enabling him to act early in the disease and under the best possible conditions.

Usually certain other signs assisting in the diagnosis are present in conjunction with the hematuria.

In *incipient tuberculosis of the kidney*, there are present certain mild vesical symptoms, such as frequent urination, a light pulverulent deposit at the bottom of the chamber, and slight pain in the bladder, and sometimes enlargement and sensitiveness of the kidney are also present (see *Examination of the Kidney. Renal points of tenderness*, p 335)

In *more advanced tuberculosis of the kidney*, there are present polyuria, pyuria, a more or less remote history of cystitis, and enlargement of the kidney.

In *renal calculus*, hematuria often appears abruptly, without any previous attack of pain Some kidney stones induce absolutely no pain. In other instances, however, pain is a prominent feature (dull pain in the kidney region, renal colic, and expulsion of stones). Finally, renal hematuria of calculous origin is *often* (but not always) *brought on* by fatigue, walking, horseback riding, etc

In *cancer of the kidney*, hematuria may appear very early and be the only symptom. At a later stage, however, it occurs in conjunction with pain, enlargement of the kidney, and cachexia

Apart from these three main causes of renal hematuria, there is one other which is of relatively frequent occurrence, *viz.*, *hematuric nephritis, with or without pain* This form of nephritis is

sometimes associated with only trifling general manifestations; and the diagnosis is made chiefly by ureteral catheterization; upon examination of the separated urines, the hemorrhage is found to be often bilateral (either concomitantly or alternately on the right and left), the kidneys exhibit impaired functional power, and casts are observed The pain in this form of nephritis often disappears after renal decapsulation

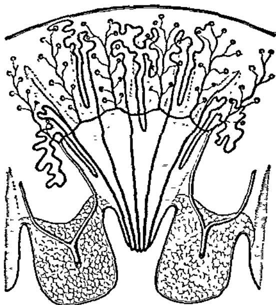


Fig 733—Diagram of the structure of the kidney *Shaded gray*, a renal pyramid, with its apex dipping into a calyx, and its base sending several processes (the pyramids of Ferrein) into the cortical substance (white) *In relief*, the arterial vessels (interlobar artery, arterial arcade, and interlobular and glomerular arteries). *In black*, a few uriniferous tubules, the course of which is from glomeruli to the apex of the renal pyramid (*Laederich*).

IV **The hematuria is of hemic origin.**—The appearance of the urine in this event is naturally the same as in hematuria of renal origin, but the cause of the condition has less to do with changes in the kidneys, which are nearly always present, than with a blood dyscrasia of infectious or toxic origin, such as:

1. *Hemorrhage-provoking disorders* of the type of *purpura, hemophilia, scurvy*, and the *leukemias*. Recently there have been re-

ported instances of actual "vesical purpura," either in conjunction with purpura of the skin or even occurring independently.

2. *Hemorrhagic forms of various infectious diseases, such as typhoid fever, malaria, small-pox, typhus fever, yellow fever, septicemia,* and *icterohemorrhagic spirochetosis.*

3. *Certain forms of poisoning associated with hemorrhage,* such as cantharis poisoning (blistering) and phosphorus poisoning (an exceptional condition aside from specialized industrial occupations).

Separate recognition must also be given to hematuria of parasitic origin of the type of *bilharziasis,* a condition met with exclusively in tropical countries or in individuals coming from such

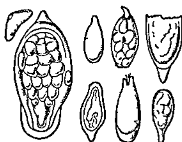


Fig 734—Bilharzia ova.

countries. The finding of the parasitic ova in the urine will alone suffice for diagnostic purposes.

Finally there remain the cryptogenic hematurias of unknown origin, including vicarious hematuria, occurring in place of the menstrual flow; the diathetic hematurias, renal "epistaxis," etc.

* * *

In any case of total hematuria, the physician must strenuously endeavor to find out the source and the cause of the bleeding.

*Cystoscopy* will of itself generally permit of establishing the origin of a vesical hematuria.

*Catheterization of the ureters,* by enabling the practitioner to collect separately the urine from the two kidneys, will give an idea of the actual functional capacity of each kidney, permit of cytologic and bacteriologic studies of the individual urines, and lead accurately to discovery of the cause of the bleeding.

*Radiography* is likewise of considerable service.

### FALSE HEMATURIA.

Mention should also be made of the *false* or *pseudo-hematurias,* in which the urine is colored red by one of a number of different drugs.

*Red discoloration of the urine by various drugs (pseudo-bloody urine).*[1]

| | |
|---|---|
| **Antipyrin:** | Blood-red color |
| **Pyramidon:** | Cherry-red or salmon color. |
| **Sulphonmethanum:** | Brownish red color |
| **Phenol and its Salts.** | Reddish brown color |
| **Cryogenin:** | Reddish dark yellow with a species of iridescence. |
| **Cascara:** | Reddish yellow or brown (red if urine is alkaline) |
| **Senna:** | Reddish yellow or brown (red if urine is alkaline) |
| **Rhubarb:** | Reddish yellow or brown (red if urine is alkaline) |

* * *

For clinical purposes, the commonest causes of hematuria appear to be those given in the following table:

[1] *After* JACQUOT *Union pharmaceutique,* 1916

## HEMATURIA.

| | History | Local Symptoms | General Symptoms | Urine | Cystoscopy |
|---|---|---|---|---|---|
| **Acute nephritis.** | Recent infection (*e g*, scarlet fever) or poisoning (*e g*, by mercury) | Lumbar pain. | Fever Headache Vomiting Edema High blood-pressure | Hematuria of renal type Albuminuria. Blood casts | |
| **Chronic nephritis.** | Former infection or poisoning Lead poisoning | Lumbar pain. | No fever Headache Vomiting High blood-pressure Cardiac hypertrophy | Albumin scanty or absent. Few casts. Rarely erythrocytes | |
| **Calculous pyelonephritis.** | History of attacks of renal colic. | Tenderness over the kidney on one side Ureteral and urethral points of tenderness on the same side | Pains of renal type | Hematuria frequently coexisting with the pains Pus in the urine. | Separation of the urine points to the side involved |
| **Renal tumor.** | Often negative | Sometimes *nil* Sometimes lumbar pain and appreciable enlargement of kidney | Progressive cachexia | Spontaneous, abundant, and recurring hematuria. | Unilateral hematuria |
| **Renal tuberculosis.** | Indications of tuberculosis in personal or family history. | Frequent urination Pain in the bladder. Tenesmus | Fever Progressive cachexia. | Slight hematuria. Pus in the urine Positive guinea-pig inoculation | Unilateral pyuria. Often ecchymoses and ulcerations of the bladder |

| | | | | | |
|---|---|---|---|---|---|
| **Acute and subacute cystitis.** | Gonorrhea. Catheterization Prostatic hypertrophy Stricture | *Frequent urination.* Pain in the bladder Tenesmus. | Slight or absent. | Pus in the urine Slight hematuria. Bladder cells | Inflamed bladder. |
| **Tumor of the bladder.** | History of attacks of hematuria. | Frequently absent. | Anemia. | Recurring hematuria | *Tumor of the bladder.* |
| **Acute urethritis.** | Urethritis. | Pain on urination. Urethral discharge. | Slight or absent. | Slight hematuric attacks of urethral type | *Negative, unless* there are bladder complications. |
| **Dyscrasic hematuria.** | 1 Purpura or hemophilia 2 Infections, such as malaria, infectious jaundice, etc 3 Intoxication | Total hematuria or hemoglobinuria Usually albuminuria | Those of the causative disorder 1 Purpura, hemophilia, or the hemorrhagic diathesis 2 Typhoid state, jaundice, hemorrhage (grave icterus) 3 Intoxication. | Total hematuria. *Hemoglobinuria* Usually albuminuria | Negative *Sometimes* "purpuric spots" in the bladder. |
| **Cryptogenic hematurias** | Sometimes as vicarious menstruation | Sometimes renal or *ureteral pain due to* clots | | Hematuria | |

**A definite, essential rule, which should be kept in mind as axiomatic, is that any case of total hematuria believed to be of vesical origin demands examination of the bladder by cystoscopy and rectovesical palpation.**

## HEMATURIA.

| | History | Local Symptoms | General Symptoms | Urine | Cystoscopy. |
|---|---|---|---|---|---|
| **Acute nephritis.** | Recent infection (*e g*, scarlet fever) or poisoning (*e g*, by mercury) | Lumbar *pain* | *Fever* *Headache*<br>Vomiting<br>Edema<br>High blood-pressure | *Hematuria of renal type*<br>Albuminuria<br>Blood casts | |
| **Chronic nephritis.** | Former infection or poisoning. Lead poisoning | Lumbar pain. | No fever<br>Headache Vomiting<br>High blood-pressure<br>Cardiac hypertrophy | Albumin scanty or absent.<br>Few casts.<br>Rarely erythrocytes | |
| **Calculous pyelo-nephritis.** | History of attacks of renal colic. | Tenderness over the kidney on one side<br>Ureteral and urethral points of tenderness on the same side | Pains of renal *type*. | *Hematuria frequently* coexisting with the pains<br>Pus in the urine. | *Separation of the* urine points to the side involved. |
| **Renal *tumor.*** | Often negative. | Sometimes *nil*<br>Sometimes lumbar pain and appreciable enlargement of kidney. | Progressive cachexia. | Spontaneous, *abundant*, and recurring hematuria | *Unilateral hematuria.* |
| **Renal tuberculosis.** | Indications of tuberculosis *in personal* or family history. | Frequent urination<br>Pain in the bladder<br>Tenesmus | Fever.<br>Progressive cachexia. | Slight hematuria.<br>Pus in the urine.<br>Positive guinea-pig inoculation. | Unilateral *pyuria.*<br>Often ecchymoses and ulcerations of the bladder |

*Hemiplegia of cortical origin (cerebral softening):*

Hemiplegia complete or incomplete.

Aphasia frequently present.

Tendency to recession of paralysis in the days following the stroke.

## Hemiplegia of Cerebral Origin.

**Paralysis of the lower branch of the facial and of the extremities on the side opposite the lesion.**

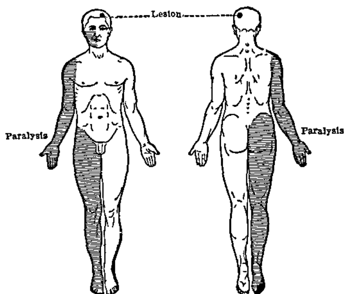



Fig 735

*Hemiplegia of capsular origin (cerebral hemorrhage):*

Hemiplegia complete and permanent.

No aphasia.

Tendency to extension in the days following the stroke.

Initial reduction of body temperature.

Early contractures

*Conjugate deviation of the head and eyeballs.*

More frequent involvement of the right side.

(These differential features, however, are not absolutely reliable).

B. Paralysis of mesocephalic origin, *relatively uncommon,* occurs in the form of an *alternate (crossed) hemiplegia,* the extremi-

# HEMIPLEGIA.

[*ἥμισυς, half; πλήγειν, to strike; more or less complete loss of motion in one side of the body.*]

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The term *hemiplegia* refers to a paralytic condition restricted to one lateral half of the body, right or left. Hemiplegia may be *complete* or *incomplete* according to the degree of paralysis. As a rule its presence is very readily observed

*For practical purposes the diagnosis of hemiplegia necessarily* entails an answer to each of the two following questions:

1 Where is the lesion located?
2. What is its nature?

**I. Location of the lesion.**—The site of disease, usually located at some point along the pyramidal tract, may be either cerebral (cortical or capsular), mesocephalic (peduncular or pontine), bulbar, or spinal Clinically, hemiplegia of cerebral origin is far more frequent than all other varieties combined. These various *types of hemiplegia are rather easily distinguished by virtue of* the following features:

A. **Paralysis of cerebral origin** is of the *hemiplegic* type, involving more or less completely the face and the upper and lower extremities. It usually sets in with an apoplectic attack; is sometimes associated *with conjugate deviation of the head and eye*-balls, and spares the muscles having synergistic bilateral motor functions.

The sensory functions are seldom affected; the tendon-reflexes are, as a rule, exaggerated; there is inversion of the plantar reflex (see *Reflexes Babinski's sign*). *Muscular atrophy is never present* at the outset and seldom later On the contrary, secondary contracture frequently occurs at the end of two or three months.

**Cortical hemiplegia,** usually induced by *cerebral softening* as a result of *embolism* or *thrombosis,* is distinguished from *capsular hemiplegia,* the result of *cerebral hemorrhage,* by the following features:

2. Alternate hemiplegia of pontine (*lower pontine*) origin results in the so-called *syndrome of Millard-Gubler*, with paralysis of the limbs on the side opposite to that of the brain lesion and paralysis of the face and internal oculomotor on the same side. As shown in the annexed illustration:

(*a*) The lesion of the pyramidal tract before its decussation results in paralysis of the extremities on the side opposite to that of the lesion

(*b*) The lesion of the facial or 7th cranial nerve at its origin results in paralysis of the face on the same side as the brain lesion

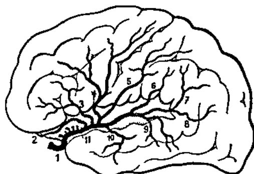



Fig 737 —Branches of the middle cerebral artery Cerebral softening *1* Middle cerebral artery *2* External orbital artery *3* Inferior frontal artery *4* Ascending frontal artery, *5* Ascending parietal artery *6.* Inferior parietal artery *7* Artery to angular gyrus *8.* Temporal arteries *9* Near the origin of the Sylvian artery are to be seen the perforating (*anterolateral*) *arteries*

It should be noted that in embolism (as by a piece of valve, vegetation, or clot), the occluding foreign body rarely enters the innominate artery, which opens obliquely into the arch of the aorta, but nearly always passes into the left carotid, which forms an almost direct prolongation of the aortic arch Thus, hemiplegia due to embolism is almost always a right-sided hemiplegia, due to a lesion of the left cerebral hemisphere.

(*c*) The lesion of the external oculomotor (abducens) or 6th cranial nerve at its origin results in paralysis of the external rectus muscle on the side of the brain lesion, *i e.*, clinically in convergent strabismus with homonymous diplopia (displaced image on the same side).

The *aphasia bundle* passes down from Broca's convolution to the anterior portion of the internal capsule and from there to the bulbopontine centers of articulate speech production.

ties being paralyzed on one side and the face or eyeball on the other.

1. **Alternate hemiplegia of peduncular** (*upper pontine*) **origin** results in the so-called *syndrome of Weber*, with paralysis of the face and limbs on the side opposite to that of the brain lesion, and oculomotor paralysis on the same side. As shown in the annexed illustration:

**Anatomic Features.**

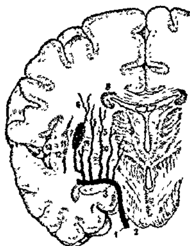


FIG 736—Striate arteries—capsular hemorrhage *1* Internal carotid *2* Anterior cerebral artery *3* Middle cerebral artery coursing through the Sylvian fissure *4*. Internal striate arteries. *5* External striate arteries *6* The artery of cerebral hemorrhage, with a miliary aneurysm in its course *7* Cerebral hemorrhage *8* Caudate nucleus *9* Optic thalamus. *10* Internal capsule. *11* Claustrum *12* External capsule *13*. *Lobule of the insula. 14 Lenticular nucleus or extraventricular nucleus* of the corpus striatum.

(*a*) The lesion of the pyramidal tract and geniculate fibers before their decussation results in paralysis of the face and extremities on the side opposite to that of the lesion.

(*b*) The lesion of the oculomotor or 3d cranial nerve at its origin causes, on the same side as the brain lesion, paralysis of the superior rectus, inferior rectus, internal rectus, inferior oblique, pupillo-constrictor, and levator palpebræ muscles, such paralyses, in turn, resulting clinically in blepharoptosis, mydriasis, divergent strabismus, paralysis of accommodation, and crossed horizontal diplopia.

2. Alternate hemiplegia of pontine (*lower pontine*) origin results in the so-called *syndrome of Millard-Gubler*, with paralysis of the limbs on the side opposite to that of the brain lesion and paralysis of the face and internal oculomotor on the same side. As shown in the annexed illustration:

(*a*) The lesion of the pyramidal tract before its decussation results in paralysis of the extremities on the side opposite to that of the lesion

(*b*) The lesion of the facial or 7th cranial nerve at its origin results in paralysis of the face on the same side as the brain lesion.

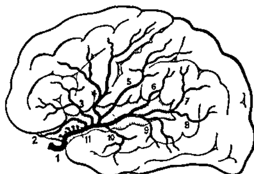



Fig 737 —Branches of the middle cerebral artery Cerebral softening *1* Middle cerebral artery *2* External orbital artery *3* Inferior frontal artery *4* Ascending frontal artery, *5* Ascending parietal artery *6*. Inferior parietal artery *7* Artery to angular gyrus *8* Temporal arteries *9* Near the origin of the Sylvian artery are to be seen the perforating (anterolateral) arteries

It should be noted that in embolism (as by a piece of valve, vegetation, or clot), the occluding foreign body rarely enters the innominate artery, which opens obliquely into the arch of the aorta, but nearly always passes into the left carotid, which forms an almost direct prolongation of the aortic arch Thus, hemiplegia due to embolism is almost always a right-sided hemiplegia, due to a lesion of the left cerebral hemisphere

(*c*) The lesion of the external oculomotor (abducens) or 6th cranial nerve at its origin results in paralysis of the external rectus muscle on the side of the brain lesion, *i e*, clinically in convergent strabismus with homonymous diplopia (displaced image on the same side).

The *aphasia bundle* passes down from Broca's convolution to the anterior portion of the internal capsule and from there to the bulbopontine centers of articulate speech production.

The *geniculate bundle* passes down from the foot of the ascending frontal convolution, passes through the internal capsule at the genu, decussates at the pons (see below), and terminates in the centers of the facial and hypoglossal nerves.

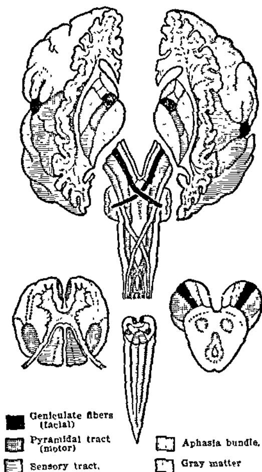



Fig 738.—Diagram of the motor and sensory paths in the brain and spinal cord

The *pyramidal tract* passes down from the central convolutions, passes through the internal capsule in its posterior and middle portion, behind the geniculate ganglion, and decussates with the pyramidal tract of the opposite side at the bulbar pyramids, after giving off a direct bundle which remains in the corresponding half of the cord.

## Peduncular hemorrhage.

**Weber's syndrome or superior alternate hemiplegia.**

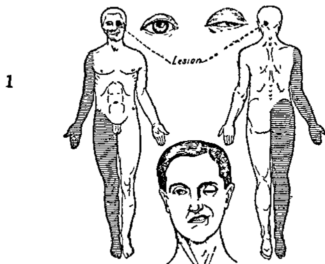



Fig 739—*1* Paralysis of the extremities on the side opposite the brain lesion *2* Oculomotor paralysis on the same side as the lesion (*a*) Ptosis (*b*) Outward deviation of the eye owing to persistence of function of the fourth and sixth cranial nerves

## Inferior pontine hemorrhage.

**Syndrome of Millard-Gubler. Inferior alternate hemiplegia.**

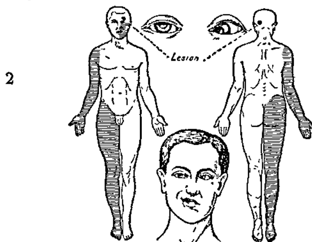



Fig 740—*1* Paralysis of the extremities on the side opposite the brain lesion *2* Paralysis of the face on the same side as the lesion *3* Paralysis of the sixth cranial nerve on the side of the lesion, causing convergent strabismus through deviation of the eye inward and downward

## Anatomic Relations.

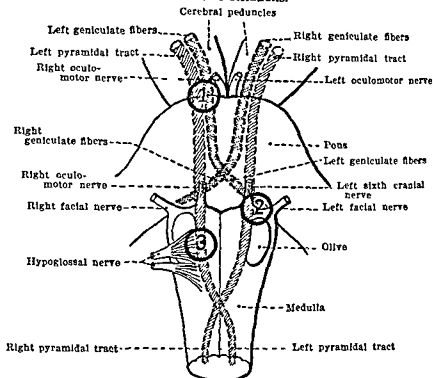



Fig 741.

### 1. High lesion involving peduncle and pons.

Superior alternate hemiplegia.

1 Involvement of the pyramidal tract above its decussation: Paralysis of the extremities on the side opposite the lesion.

2 Involvement of the facial above its decussation: Facial paralysis on the side opposite the lesion

3 Involvement of the oculomotor on the side of the lesion Oculomotor paralysis on the side of the lesion (superior rectus, inferior rectus, internal rectus, inferior oblique, constrictor of the pupil, and levator palpebræ superioris), whence

Blepharoptosis, mydriasis, paralysis of accommodation, divergent strabismus, and crossed horizontal diplopia

### 2. Inferior pontine lesion.

Inferior alternate hemiplegia.

1 Involvement of the pyramidal tract above its decussation: Paralysis of the extremities on the side opposite the lesion.

2 Involvement of the facial below its decussation Facial paralysis on the side of the lesion

3 Involvement of the abducens at its point of origin: Paralysis of the external rectus on the side of the lesion, whence

Convergent squint with homonymous diplopia (false image on same side)

### 3. Bulbar lesion (very rare).

1 [illegible] glossal and the

Paralysis of

[illegible] ralysis of the

3

**Bulbar lesion.**

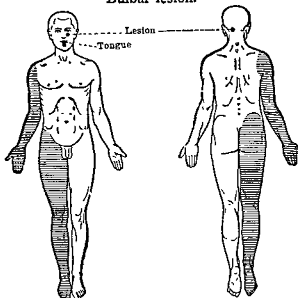



Fig 742—*1* Paralysis of the extremities on one side. *2*. Paralysis of the tongue on the opposite side

**Section of the cervical cord (extremely rare).**

**Spinal hemiplegia.**

**Brown-Séquard's syndrome.**

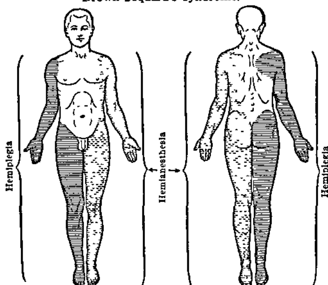



Fig. 743—*1*. Paralysis of the extremities on the side of the lesion *2*. Hemianesthesia on the side opposite the lesion *3* Face unaffected.

The *sensory tract* originating from the posterior columns of the spinal cord ascends to the bulb, where it decussates at the posterior portion of the cerebral peduncles, passes through the internal capsule in its posterior part, and terminates in the occipital convolutions.

C. Hemiplegia of bulbar origin is *exceedingly uncommon.*

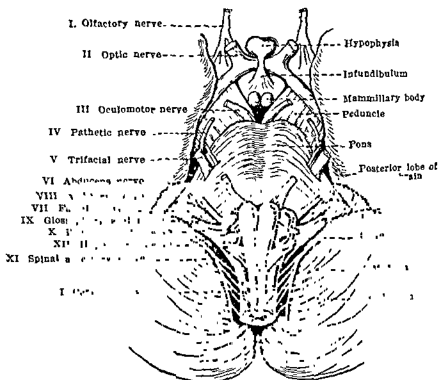



Fig 744—The medulla and pons.

The commonest example of superior bulbar paralysis is that attending *polioencephalomyelitis,* the cardinal syndrome of which is external ophthalmoplegia, *i e*, paralysis of all the muscles of the eye except the pupillary muscles

The commonest example of inferior bulbar paralysis is that of *labioglossolaryngeal paralysis,* attended with paralysis and atrophy of the lips, tongue, muscles of mastication, soft palate, and laryngeal muscles, resulting in progressive disturbances of deglutition, respiration, and circulation

(*a*) The lesion of the pyramidal tract before its decussation (see p. 1020) results in paralysis of the extremities of the opposite side.

(*b*) The lesion of the hypoglossal nerve at its origin causes paralysis of the tongue on the same side as the lesion

D. **Hemiplegia of spinal origin**, a condition *wholly exceptional* in the absence of traumatic injury of the cervical cord, results in the so-called *syndrome of Brown-Séquard*, in which there is paralysis of the limbs on the side opposite to that of the lesion, anesthesia likewise of the opposite side, and the face and eyes uninvolved.

E. Finally, one should not overlook the possibility of a purely functional hemiplegia, independent of any nervous lesion.

**Hysterical hemiplegia** is differentiated from the organic hemiplegias already described by the following features

Differential Diagnosis.

| Organic Hemiplegia | Hysterical Hemiplegia |
|---|---|
| The tendon and skin reflexes are affected.<br>There is exaggeration of the patellar reflex, abolition or diminution of the skin reflexes, and inversion of the plantar reflex (toe phenomenon or Babinski sign) | The tendon and skin reflexes are unchanged The toe phenomenon (Babinski sign) is absent |
| The disturbances of sensation decrease as one ascends from the distal to the proximal portions of the extremities | The anesthesia or hypoesthesia present is of the hemianesthetic type |
| | There is usually no facial paralysis, although there is sometimes glossolabial spasm on the opposite side. |
| The patient is suffering from heart disease, arteriosclerosis, syphilis, uremia, or other condition | The patient is generally young, most often of the female sex, and exhibits stigmata of hysteria and neuropathic evidences<br>The syndrome appears abruptly, frequently after some emotional impression. |

The *sensory tract* originating from the posterior columns of the spinal cord ascends to the bulb, where it decussates at the posterior portion of the cerebral peduncles, passes through the internal capsule in its posterior part, and terminates in the occipital convolutions

C. Hemiplegia of bulbar origin is *exceedingly uncommon.*

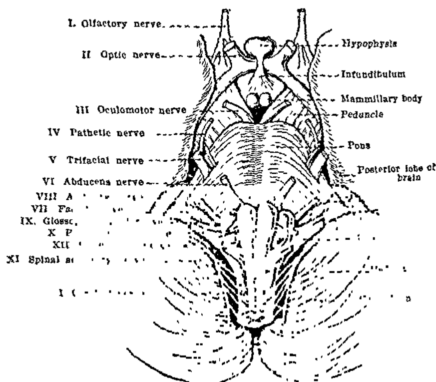



Fig 744—The medulla and pons.

The commonest example of superior bulbar paralysis is that attending *polioencephalomyelitis,* the cardinal syndrome of which is external ophthalmoplegia, *i.e.,* paralysis of all the muscles of the eye except the pupillary muscles.

The commonest example of inferior bulbar paralysis is that of *labioglossolaryngeal paralysis,* attended with paralysis and atrophy of the lips, tongue, muscles of mastication, soft palate, and laryngeal muscles, resulting in progressive disturbances of deglutition, respiration, and circulation.

(*b*) A right- or left- sided hemiplegia, appearing after an apoplectic stroke in a middle-aged or old subject is generally dependent upon *cerebral hemorrhage,* the commonest causes of which are *Bright's disease* and *arteriosclerosis.*

The predisposing influence of *plethora* in males and of the *menopause* in women may here be noted.

(*c*) An incomplete right-sided hemiplegia, appearing abruptly, without a stroke, in a young or adult subject suffering from mitral or mitro-aortic endocarditis is induced by *cerebral embolism occurring as a complication of endocarditis.*

(*d*) A right-sided, progressive hemiplegia, appearing without an apoplectic stroke in a young or adult subject who is syphilitic is generally due to *syphilitic arteritis.*

(*e*) A hemiplegia appearing more or less abruptly in a young or adult subject addicted to alcoholism, and soon accompanied by contractures or even convulsive attacks of the Jacksonian type should direct the observer toward the thought of a *hemorrhagic pachymeningitis in an alcoholic individual.*

(*f*) The following brief tabular statement of the diagnostic features of *facial paralysis* should be kept constantly in mind:

Facial Paralysis.

| | | |
|---|---|---|
| Medical causes | (*a*) Peripheral | Both branches are involved, influenzal neuritis, and the ordinary so-called "rheumatic" facial paralysis. |
| | (*b*) Central | Lower branch alone involved, cerebral hemorrhage or softening, or tumor of the cortex |
| Traumatic causes | (*a*) Intratemporal | Fractured skull, foreign body in the mastoid, or mastoid operation |
| | (*b*) Extratemporal | Wound of the face by a missile or by an operation in the parotid region |

(After *Pouchet*)

6 Blood examination (blood obtained by cupping or vein puncture).

**II. What is the cause of the hemiplegia?**

Certain general features of nervous disease may, in the first place, be referred to.

The **brain** is more frequently affected by disorders of vascular origin (thrombosis or hemorrhage) than by inflammatory disorders (meningo-encephalitis).

The **spinal cord** is more frequently involved in inflammatory degenerative processes, acute (myelitis) or chronic (systematized sclerotic processes), than in vascular lesions.

The **peripheral nerves** are oftenest affected by intoxications (alcoholism, lead poisoning, etc.), infections (diphtheria, typhoid fever, etc.), and traumatic injuries.

It is impracticable and would be tiresome to review all the possible causes of hemiplegia.

Let it be borne in mind that practically *95 per cent. of all hemiplegias are dependent upon arteriosclerosis, Bright's disease, syphilis, alcoholism, or rheumatism (endocarditis)*, and that these several causes can be almost always discovered by the following plan of examination:

1. **History:** Age, vertigo, mental impairment (arteriosclerosis).

Polyuria, nycturia, albuminuria, etc. (Bright's disease)

Specific history (syphilis).

Intemperate habits (alcoholism).

Acute rheumatism or other acute infection.

2. **Auscultation:** Aortic evidences (arteriosclerosis, specific aortitis).

Aortic evidences with gallop rhythm (Bright's disease).

Mitral or mitro-aortic evidences (rheumatism).

3. **Examination of the urine:** Albuminuria and casts (Bright's disease, arteriosclerosis).

4. **Blood-pressure determination:** Pressure particularly high in arteriosclerosis and Bright's disease.

5. **Special features of the hemiplegia** *per se*

(*a*) A right-sided hemiplegia without apparent cause, after a short period of dizziness, with or without an apoplectic stroke, and without temperature changes, generally points to *softening of the brain due to thrombosis in an arteriosclerotic subject.*

**Patient a child.**

**Tuberculous meningitis**—Hemiplegia appears at an advanced stage of the disease, there is therefore a preliminary period of illness or history of pre-existing meningitis

**Meningeal hemorrhage.**—This is attended with contractures, and the cerebrospinal fluid contains blood.

**Cerebral sclerosis**—Simultaneous appearance of mental disturbances and trophic changes

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II **Hemiplegia** coming on after **manifest traumatic injury** (fall, blow on the head, gunshot wound, shell fragment, sword stroke, etc)

**Fracture of the skull.**—The history, direct examination of the wound, and direct exploration (removal of bone fragments, trephination, etc)

**Hemorrhage**—Lumbar puncture and the attendant circumstances and related symptoms supply all the factors needed for diagnosis of the location and extent of the hemorrhage—very important features in relation to surgical intervention

**Contusion of the brain**

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III **Hemiplegia** coming on in the course of **some other nervous disease.**

**General paralysis**—Here it is generally due to a concomitant cerebral hemorrhage, and presents the usual characteristics of the latter

**Tabes dorsalis.**—Here it is accompanied by paralysis of the oculomotor nerves, loss of the patellar reflexes, the Argyll-Robertson pupil, and other signs of tabes

**Hysteria.**—Here it presents the usual features of hysterical hemiplegia (Babinski sign, hemianesthesia, and other significant stigmata).

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IV. **Hemiplegia** coming on in the course of **some general disorder.**

Lead poisoning, mercurial poisoning, nephritis, uremia, arteriosclerosis, and various infectious diseases—The hemiplegia is produced in one of the ways already referred to, *viz*, thrombosis, hemorrhage, or embolism—Its features and significance are those already described.

## HEMIPLEGIA.

| CAUSES | SPECIAL FEATURES | HISTORY. | ASSOCIATED SIGNS. | COURSE. | LUMBAR PUNCTURE. |
|---|---|---|---|---|---|
| **I. Hemiplegia** coming on more or less **abruptly** is the main dominant manifestation of the illness. (If the patient is already in the stage of coma, see *Coma*). | | | | | |
| **Patient an adult.** | | | | | |
| **Softening following thrombosis.** | Hemiplegia appearing after a period of dizziness, with or without an apoplectic attack, sometimes gradually<br>Greater frequency on the right side | Patient old, sometimes with previous gradual mental impairment.<br>Arterial sclerosis | No temperature disturbance at the start | Tendency to partial subsidence | |
| **Cerebral hemorrhage.** | Aphasia frequent<br>Abrupt hemiplegia after an apoplectic attack<br>As frequent on the left side as on the right<br>No aphasia | Patient adult or old<br>Plethoric or alcoholic<br>Gouty or nephritic<br>Menopause or atheroma | Initial lowering of temperature<br>Later rise in the event of fatal termination | Tendency to extension on the days following the stroke<br>Early *contractures*. | Red cells in the cerebrospinal fluid. |
| **Cerebral embolism.** | *Abrupt hemiplegia*, generally incomplete, without apoplectic stroke, more frequent on the right side | Subject young or adult<br>History of rheumatism | Presence of endocarditis | Tendency to rapid subsidence | |
| **Syphilitic arteritis.** | Similar to hemiplegia due to thrombosis | Subject young or adult.<br>History of syphilis<br>Often Wassermann positive | | Rapid improvement under specific treatment | Wassermann positive |
| **Hysteria.** | Hemiplegia and hemianesthesia<br>No disturbance of reflexes | Subject young<br>Sudden onset after emotion | Neuropathic stigmata | | |

Due thought should be given to **exceptional conditions**, such as tuberculosis of the brain (general condition, chest evidences, and fever); meningeal hemorrhage and hemorrhagic pachymeningitis (*alcoholic subject, with Jacksonian epilepsy*)—Still more exceptionally· Cervical Pott's disease, hemisection of the cervical cord (Brown-Sequard's syndrome), special localization of an acute poliomyelitis

# HEMOPTYSIS. [αἷμα, *blood*; πτύσις, *spitting*. *Spitting of blood*. (See *Expectoration*.)]

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**Hemoptysis,** defined as the expectoration of blood set free in the air passages, is very often an obvious occurrence, yet rather frequently the fact that the blood expectorated has come from the broncho-pulmonary tract requires to be investigated, verified, and proven Hemoptysis and hematemesis are often confused, and even more frequently, more or less deeply blood-stained discharges of nasal, lingual, gingival, or laryngeal origin are put down as constituting hemoptysis.

**Epistaxis.**—Production of bloody expectoration by epistaxis, especially of the posterior variety, is a very commonly observed occurrence. Where expectoration of blood attends an anterior "external" epistaxis, the source of the blood is obvious; where, however, it is not accompanied by "nosebleed," examination of the pharynx and even the nasopharynx is indicated Nasopharyngeal hemorrhage is generally of dyscrasic origin (hemophilia or high blood-pressure) or the result of ulceration, rupture of a varicose vessel, or an adenoid inflammation which careful examination cannot fail to detect

The only real, though slight, diagnostic difficulty arises from the fact that blood shed in the nose or pharynx may actually be swallowed or coagulate in the nasopharynx itself and be discharged only by a spell of coughing Under these conditions the blood is more or less mixed with mucus from the stomach or nasopharynx and may, upon hasty examination, be deemed to have issued from the lung or stomach The opposite mistake, which consists in carelessly ascribing to the nasopharynx a hemorrhage actually occurring in the stomach or lung, is much more serious

Let it be repeated once again that a careful general examination—*including that of the nasopharynx*—will inevitably and

(*a*) The *Wassermann reaction*, if positive, will confirm the diagnosis of syphilis if it has already been rendered, or will draw the observer's attention in that direction in doubtful cases.

(*b*) *Determination of the blood urea*· Blood urea exceeding 0 80 gram indicates the presence of uremia.

Much might be written concerning the relationship of uremia to hemiplegia Formerly it was practically an established custom to consider the terms relatively antagonistic. At the present time one cannot help noting that these two conditions bear most intimate clinical interrelationships, and that hemorrhages (including cerebral hemorrhage), high blood-pressure, azotemia, and albuminuria are practically common accompaniments of *arteriosclerosis* and *nephritis*. Concomitance of the two conditions is, indeed, the clinical rule. Most of the author's cases of sclerosis with hemiplegia have been azotemic, and many of his uremic subjects have shown a terminal hemiplegia Such are the actual facts in this connection.

7. **Examination of the cerebrospinal fluid.**—This is of especial service for the detection of:

1. Hemorrhagic states· Cerebral and meningeal hemorrhage (red blood cells in the cerebrospinal fluid).

2. Inflammatory states· Meningitis (leucocytosis; presence of pathogenic bacteria).

The annexed clinical table summarizes the facts already recalled.

# HEMOPTYSIS. [αἷμα, *blood*; πτύσις, *spitting. Spitting of blood.* (See *Expectoration*.)]

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**Hemoptysis,** defined as the expectoration of blood set free in the air passages, is very often an obvious occurrence; yet rather frequently the fact that the blood expectorated has come from the broncho-pulmonary tract requires to be investigated, verified, and proven Hemoptysis and hematemesis are often confused, and even more frequently, more or less deeply blood-stained discharges of nasal, lingual, gingival, or laryngeal origin are put down as constituting hemoptysis.

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Let it be repeated once again that a careful general examination—*including that of the nasopharynx*—will inevitably and

promptly lead to a correct determination of the source of expectorated blood.

The **lingual origin** of blood expelled from the mouth may be noted in the event of a bite of the tongue, traumatic or "epileptic," or of a tuberculous or neoplastic ulceration. Mention of this cause of blood spitting, which could confuse *only* a very careless or inexperienced practitioner, is required merely for the sake of completeness and because it is customary to do so.

**Bleeding from the gums** is extremely common, even independently of scurvy and hemophilia, which are themselves exceptional disorders. The ubiquitous pyorrhea alveolaris, various *diathetic disturbances, and even abuse of the tooth brush may* induce swelling and "inflammation" of the gums and render them both sensitive and prone to bleed. Under these circumstances the least contact with the gums results in oozing which stains all sputum with blood and gives rise to "pseudo-hemoptysis." A most casual examination of the gums will discover the *actual source of the bleeding to be in these structures.*

**Hemorrhage in the larynx** may result either from traumatism or from ulceration The former is readily excluded Ulcerations are always either syphilitic, tuberculous, or cancerous; they are always attended with hoarseness and local pain; they are always preceded by a period during which the attention was drawn to *and fixed upon the larynx. Examination of the larynx with the* mirror will always demonstrate the laryngeal source of the bleeding.

The only real difficulty in diagnosis is that sometimes met with in the **differentiation of hemoptysis from hematemesis,** which may constitute a most puzzling problem (see *Hematemesis*).

*As a rule, there is little trouble in this connection:*

The *preliminary symptoms* are different, being

Digestive in hematemesis and respiratory in hemoptysis

The *manner in which the blood is expelled* is likewise different

In hemoptysis there are attempts at coughing and the blood is liquid, red, and foamy.

In hematemesis there is retching and the blood is clotted, dark colored, without admixture of air, but mixed with food material; sometimes intestinal hemorrhage coexists.

*All these signs, however, are unreliable.*

In hemoptysis there may be dark colored blood (pulmonary hemorrhage) and in hematemesis red blood (gastric ulcer).

In hemoptysis the preliminary cough may be wanting, while on the other hand there may be concomitant vomiting and consequently admixture of blood with food material.

In hematemesis the blood may or may not be mixed with bile or food material.

After careful deliberation and thorough examination the conclusion may eventually be reached that hemoptysis has occurred.

* * *

The diagnosis of "true hemoptysis" having been correctly made —and definitely established, *i e.*, on the whole, the presence of blood in the sputum either being obvious or having been demonstrated by suitable hematologic procedures; the supposition of hematemesis having been excluded, sometimes a difficult matter, as the author has seen mistakes in this connection made (and later proven) by experienced observers; and the oropharynx, gums, tongue, and nasopharynx being readily eliminated as sources of bleeding by a mere cursory examination, *provided it is actually carried out*,—the *cause* of the hemoptysis is generally very easily found if the following fundamental propositions are kept in mind:

1. Eleven-twelfths of all instances of true hemoptysis are of cardiopulmonary origin and are due to one of two following causes

(*a*) Pulmonary tuberculosis in any one of its stages, from the pretuberculous congestive stage to the stage of cavity production.

(*b*) Infarction of the lung, generally secondary either to a *mitral disorder*, particularly *mitral stenosis;* to some *cardio-arterial disorder* which has advanced to the stage of decompensation with stasis, or to *phlebitis* in any of its stages, from the pre-obstructive stage to the stage of disintegration of the intravascular clot.

*Pulmonary tuberculosis* is a far more frequent cause than *infarct of the lung*. As for the latter, its cause is nearly always obvious upon any sort of careful examination (mitral stenosis, heart failure, puerperal, infectious, or post-operative phlebitis, etc.);

frequently, moreover, infarction is marked by a sudden pain in the side with cough, dyspnea, and even orthopnea, an area of fine râles, etc.

Thus, any case of hemoptysis unattended with cardiovascular disease (heart disease or phlebitis) may be considered to be in all likelihood tuberculous. Furthermore, the hemoptysis of advanced tuberculosis, in the stages of softening or cavity formation, cannot fail to be recognized, as its source is quite obvious.

Doubt may arise only in connection with the premonitory

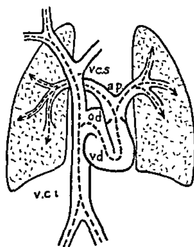



Fig. 745.—Diagram of pulmonary infarction. The embolus, having become detached at some point in the inferior vena cava (*v c i*) or its tributaries, or in the superior vena cava (*v c s.*) or its tributaries, passes through the right auricle (*o d*), is discharged into the pulmonary artery (*a p*), and lodges in one of the lobes of the lung, giving rise to an infarct which finds its clinical expression in 1 A sudden sharp pain in the side 2 Blood-spitting (hemoptysis) 3. The physical signs shown in Figs 746 and 747

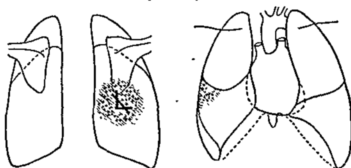
*hemoptysis at the very beginning of tuberculosis,* accompanied by little in the way of general or auscultatory evidences, or even occurring at a time when the disease is as yet entirely latent Any true hemoptysis of obscure, cryptogenic origin should be considered of tuberculous nature until proof to the contrary is obtained, and the patient kept under careful observation as regards body weight, temperature, general health, and examination of the lungs; this is a clinical axiom from which the practitioner should never depart, lest he take upon himself a most

serious responsibility and meet with egregious disappointment.

The only real difficulty in these cases lies in the possibility of simultaneous mitral stenosis and pulmonary tuberculosis; only rarely, however, will careful observation of the patient's general condition and temperature and systematic, repeated auscultation of the lungs fail sooner or later to afford a distinction between the manifestations appertaining to one or the other of these disorders

Certain instances of hemoptysis, due exclusively to mitral stenosis and associated with marked and persistent hyperemia of

Physical signs.



Weakened or muffled breathing
Surrounding area of crepitant râles

Figs 746 and 747—Pulmonary infarction

a pulmonary apex, may, however, hold the diagnosis in suspense for a time.

2 One twelfth of all instances of true hemoptysis may be referable to exceptional or obvious or, on the other hand, with difficulty detected causes.

Among the *obvious causes, traumatism* is the most important; thus, a chest wound or contusion, a fractured rib, or gas poisoning are obvious conditions.

The majority of acute and, particularly, chronic affections of the lungs may ultimately be associated with hemoptysis Accordingly, hemoptysis may be met with in pneumonia, chronic bronchitis with dilated bronchi, abscess of the lung, broncho-

pulmonary gangrene, and syphilitic or cancerous involvement. Frequently the clinical evidences present in conjunction with the hemoptysis are such, especially in bronchiectasis and pulmonary abscess or gangrene, as to make the diagnosis perfectly plain; syphilis and cancer, however, require a painstaking examination for their detection, and especially, the actual thought of their possible presence on the part of the physician.

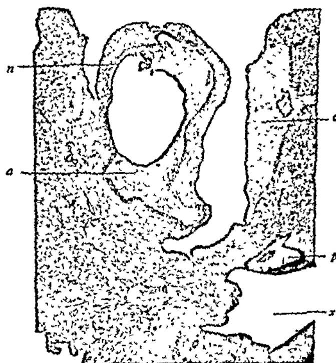



*Fig 748.—Aneurysm of the pulmonary artery (Letulle and Nattan-Larrier Microphotogr by E. Normand ×5) n, ruptured wall of the affected artery, c, tuberculous cavity bounded by a layer of caseous material, x, a diverticulum of the aforesaid cavity, p, pulmonary vein, occluded and included in the caseous layer about the cavity.*

**Hemorrhagic disorders**, including the infectious purpuras, infectious jaundice, hemophilia, leukemic conditions, etc, as well as some severe forms of typhoid fever and malaria, may likewise lead to hemoptysis, but in these disorders the hemorrhage takes place in the presence of a clinical picture so definite as to leave little chance of any actual difficulty in diagnosis. One of the author's patients with arteriosclerosis and high blood-pressure

developed every spring, in April and May, for over ten years, a varied assortment of hemorrhages, usually in the form of epistaxis, sometimes as hemoptysis and less frequently as hemorrhoidal hemorrhages of almost alarming extent; the tenth year, he developed a severe cerebral hemorrhage resulting in permanent hemiplegia.

In addition to these hemorrhagic disorders mention should be made of the *congestive hemoptysic attacks of gouty patients;* the author has seen several instances of this condition The history of gout, the congestive nature of the pulmonary attack—which gen-

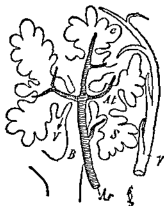



Fig 749—Diagram of a pulmonary lobule (*Miller*) *B*, terminal bronchus, *V*, vestibule, *At*, atrium, *S*, air-sac (infundibulum), *C*, air-cell (alveolus), *Ar*, artery, *V* (at the right), vein

erally involves the bases of the lungs and sometimes assumes the appearances of acute edema—and the overnourished aspect of the patient directly suggest the diagnosis in such cases. *Uremia* may give rise to a similar condition, the source of which is ascertained by blood-pressure estimation and determination of the blood urea

Aortic aneurysm may likewise lead to hemoptysis, under two very different groups of circumstances There may occur either slight, intermittent hemoptysis, due to the presence of a slight fissure at an area of adhesion of the aneurysm to the trachea and sometimes compatible with life for a more or less prolonged period, or else rupture of the aneurysm into a bronchus or the trachea with fatal hemorrhage From the diagnostic standpoint, either the presence of aneurysm will already have long been

known at the time of the hemoptysis, the significance of which will therefore be obvious, or the aneurysm will have up to that time remained latent, either because of insufficient clinical examination or on account of a remarkable degree of tolerance of the disease by the patient, who has not as yet consulted a physician. In no case of this type, to the author's knowledge, has careful clinical examination failed to elicit some sign or other of a mediastinal mass (bronchial murmur, collateral circulation over the upper part of the thorax, pressure manifestations, pupillary inequality, elevation or broadening of the arch of the aorta, inequality of the right and left pulse, etc ).

Few symptoms, the reader will note, are of such definite semeiologic significance as hemoptysis. Almost constantly it constitutes an external expression of some pulmonary or cardiac disorder, hemorrhagic diathesis, or manifest or latent congestive state.

**Does Vicarious Menstruation in the Form of Hemoptysis Actually Occur?**—Before concluding this section, mention may be made of a very exceptional form of hemoptysis which has been the subject of prolonged discussion and the diagnostic and prognostic significance of which may be altogether different, *viz., vicarious menstruation through the lungs.*

The reader's indulgence is requested by the author for devoting a disproportionate amount of space to this subject, but the condition is a very peculiar and as yet variously regarded one, and the following brief contribution to its study seems warranted on condition that the reader makes good note of the fact that this constitutes only a very exceptional form of hemoptysis.

According to many observers, particularly phthisiologists, vicarious menstruation through the lungs is almost invariably evidence of a manifest or threatening tuberculosis of the lungs.

The condition is, as a matter of fact, met with either singly or repeatedly in not a few cases of pulmonary tuberculosis in women But the writer has also seen it in cases in which all idea of an organic disease could be clinically excluded and in which very prolonged later observation failed to show the existence of any morbid manifestation.

One case, among others, may be here referred to because the period of observation was sufficiently prolonged to carry conviction The patient was a young lady about twenty years of age, free of any history of general disease, apparently in robust health, having one child eighteen months old and another three months old which she was nursing, and who, in November, 1901, saw her menstruation stop suddenly on the second day and a copious hemoptysis set in which continued for two days and then ceased as it had come on, without apparent cause The patient, with whom the author had been acquainted for three years, had had no discomforts during that time, her pregnancies had been normal and unattended with any noteworthy difficulties The attack of hemoptysis referred to, aside from some justifiable but temporary anxiety, was not associated with any marked disturbance; there was no rise of temperature nor of the pulse rate, nor any dyspnea. Very careful auscultation gave negative results.

Lactation was not discontinued and no special treatment was instituted Although kept under careful observation for a long period, the patient showed no further untoward symptom The author has been seeing her at somewhat irregular intervals for almost eighteen years, on account of illnesses of her husband or children, and has not seen her health in the least impaired at any time. No recurrence of the hemoptysis ever took place. It would seem difficult to supply any more positive clinical evidence than this

An almost similar clinical picture was seen by the author in a lady of thirty-five years; the ophthalmic test was negative. From the 15th to 17th of May, 1908, he had occasion to observe 6 spontaneous hemorrhages, including 2 of copious epistaxis in arteriosclerotic subjects, 1 of cerebral hemorrhage, 1 of hemoptysis in a consumptive, the case of vicarious hemorrhage already referred to, and a case of vicarious epistaxis. In this more than a mere coincidence was unquestionably involved; the subject will be taken up again in a later publication

Were the physician still to harbor any doubts as to the actual occurrence of idiopathic hemoptysis vicariously substituted for the menstrual flow, Ventura's case would certainly remove them

(*Gaz. degli osped.*, No 129, 1907). This author gave the history of a family in which substitution of periodic hemoptysis for menstruation took place in three successive generations. The first generation comprised three sisters; in one of them menstruation was replaced by hemoptysis at monthly intervals. One of the unaffected sisters had five female children, two of whom presented the same clinical phenomenon as their aunt. Again, one of the latter had four female children, two of whom showed the same inversion of function In none of these subjects was either tuberculosis, syphilis, hemophilia, or cardiac disease discovered.

Finally, as an interesting clinical case, reference may be made, although unrelated to hemoptysis, to a quite typical and remarkable instance of menstrual substitution recorded by the author at the Maison municipale de santé while in Danlos's service.

The patient was a woman about forty years of age, short and stout, who had had ten years before a child which she had nursed for a long time (up to about two years) and who had had no menstrual periods since that time but, on the other hand, had kept on secreting milk continually, with periodic recrudescences, as the author had occasion personally to observe. This woman died of a brain tumor. At the autopsy a normal uterus but atrophied uterine adnexa were found

Thus, due recognition should be given to the actual occurrence of idiopathic hemoptysis, vicariously substituted for the menstrual periods, in the absence of any organic disease in the lungs or heart and of any hemorrhagic disorder of the blood The prognostic importance of this fact is self-evident

* * *

**Bronchial Spirochetosis.**—Castellani in 1905 described a special form of bronchial infection observed in Ceylon and caused by spirochetes. In 1908, American observers reported it from the Philippines, and others, in various tropical lands Experience during the great war led to the detection of a considerable number of cases along the shores of the Adriatic and in Serbia, Switzerland, Egypt, and France The disorder is commonly mistaken for tuberculosis, whether acute or chronic in form The patients generally complain of an obstinate cough which gets worse at night and in the morning [illegible] oration In the common, chronic [illegible] y free of fever and the general [illegible] nosis of the disease is based ex- [illegible] r the organism termed by Castel- [illegible] with the basic anilin stains, but

## HICCOUGH.

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Hiccough or hiccup consists of a clonic contraction of the diaphragm. Its essential feature is a sudden inspiratory contraction of the diaphragm occurring in conjunction with a rapid closure of the glottis, there result an abdominal spasm, inspiration and sudden expulsion of air, consequent vibration of the completely or partly closed glottis, and a hiccup sound which may at times assume the characteristics of a bark.

A diagrammatic representation of the usually reflex pathogenesis of hiccough is presented below

The **medullary center** concerned adjoins that of the pneumogastric and consequently the vomiting and respiratory (cough) centers.

It appears to undergo direct stimulation, possibly through the circulation, in the course of "grave infections" and "agonic states," the resulting condition constituting a terminal form of hiccough.

The **centripetal (afferent) routes of stimulation** consist chiefly of the *pneumogastric* nerve and secondarily of the *sympathetic* and certain *cortico-bulbar* fibers

THROUGH THE PNEUMOGASTRIC (an appropriately named nerve):

The center may be brought into activity by stimuli starting:

(*a*) From the ABDOMEN (subdiaphragmatic region)

1. From the *stomach*· This is the starting-point of hiccough in 4 out of 5 cases, *e g*, very commonly in infants after nursing, in dyspepsia (especially of the neurotic type), in dilatation of the stomach, aerophagia, tachycardia, the ingestion of unduly hot or cold food, and much less frequently in ulcer and cancer of the stomach. In these cases the hiccough may be either continuous or intermittent, appearing and disappearing for no evident reason or when the subject takes food or a little fluid.

2. From the *intestine*: Helminthiasis

3. From the *peritoneum* Peritonitis, especially when situated below the liver.

4. From the *female reproductive organs:* Genitourinary and uterine disorders.

(*b*) From the THORAX (supradiaphragmatic region).

1. In *diaphragmatic pleurisy:* Actually a rare cause, the stimulus being ordinarily inhibited by pain. The same is true in pneumonia.

2. In *cardiopericardial disturbances:* Hiccough is observed especially in *pericarditis,* particularly at the beginning, when the phe-

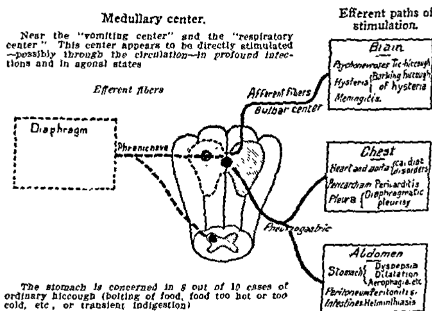



Fig 750.—Diagram illustrating the pathogenesis of hiccough

nomena of irritation are still predominant. Exceptionally the author has seen it in lesions of the aortic arch, aortitis, and aneurysm. The same is true of heart disorders, such as acute or chronic endocarditis, myocarditis, etc

*The* SYMPATHETIC ROUTE *may in all likelihood be one of the* afferent routes of stimulation of the phrenic nerve, chiefly in the presence of heart disturbances.

Finally, the *corticobulbar routes of stimulation* are those involved in *psychoneurotic states* (tic-hiccough), in *hysteria* (barking hiccough), and in *meningitis.*

The centrifugal (efferent) routes of transmission are chiefly represented by the *phrenic nerve* which is the motor nerve to the

diaphragm and the gross anatomic relationships of which should always be kept in mind Issuing from the cervical plexus, it passes down, crosses the anterior aspect of the scalenus anticus muscle, runs down along the internal border of this muscle, enters the thorax, and passes, on the right side, between the subclavian artery and vein, on the lateral aspect of the pneumo-




Fig 751—The phrenic nerves (*Hirschfeld*)

gastric, and along the superior vena cava, and on the left side, behind the innominate artery, crossing the arch of the aorta It then courses on either side between the pleura and the pericardium and ends in ramifications over the upper surface of the diaphragm

It receives the stimuli issuing from the pneumogastric:

1 Through the intermediation of the medullary center of this nerve and through the anterior gray horns

2 Through the cervical plexus (whence the phrenic nerve originates from three roots), being connected with the pneu-

mogastric through the gangliform plexus by means of one or two nerve filaments.

It is enabled to receive the stimuli from the sympathetic by reason of the many anastomotic connections between the cervical plexus and the sympathetic.

The actual cortico-bulbar pathways are not as yet satisfactorily known.

*Recognition of the cause* of hiccough is based chiefly upon the associated symptoms or physical signs, which may indicate gastric disturbance, peritoneal or meningeal involvement, psychoneurosis, etc.

During the winter of 1920 there developed, as is well known, an epidemic of hiccough in relation to which various pathogenetic conceptions were discussed, such as imitation, phrenic form of influenza, and myoclonic form of epidemic encephalitis. There is no doubt that some of the cases were of the last-mentioned origin and were accompanied by "incomplete" symptoms of encephalitis, *viz.*, slight febrile movement, myoclonic disturbances, pains, and changes in the cerebrospinal fluid. It is no less certain, however, that a majority of the cases, at least among those personally seen, were pithiatic (hysterical), and had been brought into being through the alarmist clamors of the public press.

# HIGH BLOOD-PRESSURE.

Inspection of a sphygmographic tracing of the pulse, such as that presented herewith (Fig 752), in which variations of blood-pressure within the artery under examination are elicited and recorded, is sufficient to show that this pressure is a variable quantity and that it exhibits both a high point corresponding to cardiac systole (the maximal or systolic pressure) and a low point corresponding to diastole (the minimal or diastolic pressure). There is thus not one single blood-pressure, but several

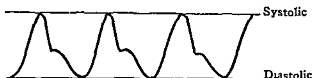



Fig 752—Pulse tracing showing the successive variations of pressure within the lumen of an artery

blood-pressures The *maximal pressure* corresponds to but a very brief portion of the cardiac cycle, the point of culmination of the systole The *minimal pressure*, on the other hand, constitutes the permanent, basic pressure below which the pressure never descends, it will be seen at once that this latter pressure is at least as important to know, and perhaps even more important, than the maximal pressure. The difference between the maximal or systolic and the minimal or diastolic pressures, the differential or pulse pressure, bears a manifest relationship to the force of the pulse and consequently to the cardiac contraction The diagram herewith presented will demonstrate at once the main features and mutual relationships of these several grades of pressure (Fig 753)

All of these three grades must be taken into account if an approximate idea of the general circulatory condition in a given subject is to be obtained

The *maximal or systolic pressure* has long been, and still is, the kind of blood-pressure investigated by the majority of cardiologists. Yet it is certainly insufficient to afford an exact idea of the circulatory balance in a given case. Convincing evidence of this is relatively easily given Following are two sphygmomanometric tracings (Fig 754) taken from the same patient, the one directly

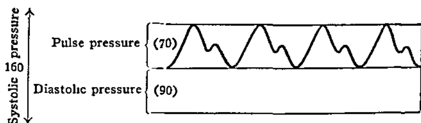



Fig 753—Diagram illustrating the systolic, diastolic, and pulse (differential) blood-pressures.

during an attack of heart failure, and the other during convalescence with a normal condition of circulatory equilibrium. The systolic and differential pressures are the same, *viz*, 200 millimeters Hg, in the two cases, but the minimal and differential pressures show, on the contrary, pronounced changes. This single example, taken from among many others, is sufficient to

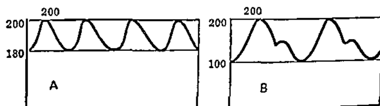



Fig 754—Blood-pressure determinations made in a single individual, a few days apart, the patient having heart failure in *A*, but restored compensation in *B*.

show that henceforth it will be necessary to be able to determine both the systolic and the diastolic pressures; this is one reason, and perhaps the most important one, which led the author to adopt the Pachon oscillometer in his blood-pressure studies

The normal systolic pressure ranges from 140 to 160 millimeters Hg (Pachon) in different individuals; exceptionally, the writer has found it 130 or 170.

This observation is in itself of considerable practical importance, showing that the concept of high blood-pressure is actually a relative or individual quantity. A person whose normal systolic blood-pressure was 130 would already be suffering from a marked increase in blood-pressure at 190 millimeters Hg; on the other hand, 190 would constitute but a moderate degree of hypertension in a patient whose normal pressure was 170.

High systolic blood-pressure starts then, in the writer's opinion, at 180 millimeters Hg.

For practical purposes three grades of high blood-pressure may be recognized

**Slight hypertension**, from 180 to 200, generally observed in *plethoric* or *full-blooded* subjects and in the *obese, gouty*, or *diabetic*, generally a result of overeating.

**Intermediate hypertension**, from 210 to 250, likewise observed in many of the *plethoric* subjects already enumerated, but also in certain number of cases of *arterial* and *renal sclerosis*

**Marked hypertension**, from 260 to 350, almost exclusively met with in *arteriosclerosis* and *interstitial nephritis*

This rough, simple nomenclature shows that the equation, high blood-pressure = arteriosclerosis, is to a large extent in error, and that, aside from *organic* (vasculorenal) *hypertension*, there occurs also a *functional* (neurohemic) *hypertension* This distinction is of decided importance, for the treatment to be instituted is quite different in the two instances

It should be borne in mind, moreover, that: (1) The systolic arterial pressure, like all other systemic coefficients (pulse rate, temperature, urinary output, etc ), exhibits normal variations of 20 to 30 millimeters Hg in the course of the day, rising from morning to evening, after meals, after exercise, etc. (2) In some sphygmolabile, angiospastic subjects sudden, temporary, and pronounced changes of pressure amounting to 40 or 50 millimeters, or even more, may be witnessed

Whence 2 practical deductions: (1) In any given subject the blood-pressure should be taken, as much as possible, at the same hour of the day, midway between meals, in order to obtain comparable readings. (2) True, continuous high blood-pressure should be considered present only after several pressure deter-

minations, made at intervals of several days, have actually shown it to exist.

The **minimal or diastolic pressure,** or constant pressure, varies within limits much more restricted than does the systolic pressure.

In the *normal subject,* it commonly ranges between 80 and 100 millimeters Hg; 80 corresponding to the normally low systolic pressures of 130 to 140 millimeters (Pachon instrument), and 100 to the normally high systolic pressures of 150 to 170 millimeters It is thus seen to be relatively stable as compared to the systolic pressure.

*Increased diastolic pressure* is met with under the following four circumstances.

1. In cases of *markedly high systolic pressure with good compensation* (260 millimeters or higher), in which the sustained diastolic pressure may reach 120 millimeters or even exceptionally 130 millimeters.

2. In cases of *heart failure,* heart failure being associated much oftener with an increase of diastolic pressure than with a reduction of systolic pressure; these two pressure indications may, indeed, coexist; in fact, they are usually present together

3. In cases of *uremia*

4. In cases of *plethora* with *venous congestion,* especially in individuals exhibiting the syndrome of *increased portal pressure*

In short, high diastolic pressure is generally associated—if, indeed, it is not actually its true sphygmomanometric expression—with high venous pressure, loss of cardiovascular balance, heart weakness, or heat failure

The highest diastolic reading the author has recorded was 190 millimeters, in a case of uremia with heart failure

Thus, the diastolic pressure is seen to be of at least as great diagnostic and prognostic significance as the systolic pressure.

Were it necessary to summarize the relative portent of high systolic and high diastolic pressure in one brief, epigrammatic, almost crude phrase, the matter might be expressed as follows:

*High systolic pressure is the balistic high-pressure having to do with rupture, hemorrhage, and apoplexy.*

*High diastolic pressure is the static high-pressure having to do with loss of cardiovascular balance, venous congestion, and heart failure.*

The differential or pulse pressure, consisting of the difference between the systolic and the diastolic pressures, commonly varies in the same direction and almost to the same extent as the systolic pressure in persons in a state of satisfactory cardiovascular balance; thus, *if absolutely necessary*, the curve of variations in the systolic pressure, practically parallel to that of the variations in the differential pressure, may be sufficient in following up the circulatory function in a subject with good circulatory balance.

In a subject on the road to heart failure, on the other hand, the differential or pulse pressure decreases much more quickly or rises much more slowly than the systolic pressure. Discrepancy is shown between the courses followed by these two kinds of pressure To estimate the progression of heart failure, the pulse pressure seems to the writer to be essential

Thus, one is led to consider the pulse pressure as a reflection or sphygmomanometric expression of cardiac power Indeed, constitutionally weak individuals with congenitally small hearts show a low pulse pressure (20 to 40 millimeters Hg); well compensated cases of arteriosclerosis with "ox hearts" show an enormously high pulse pressure (100 to 250 millimeters), cases of heart failure with low pulse pressure generally show a gradual gain in the pulse pressure as the heart recovers its functional power and the output of urine undergoes a corresponding increase, etc.

A rather absurd objection is sometimes made to this relationship of the pulse pressure to cardiac power, which at least has the advantage of bringing out in striking fashion the importance of the diastolic pressure; and, strangely enough, this objection has been raised by the very persons who have overlooked the latter variety of pressure Thus, it has been said that it is absurd to believe that any relationship exists between the pulse pressure and cardiac power Take two individuals, for example: The first shows 160 millimeters as systolic, 90 as diastolic, and 70 as pulse pressure, and is in a satisfactory state of circulatory

balance; the second shows 260 millimeters as systolic, 190 as diastolic, and again 70 as pulse pressure, but is already in a state of complete decompensation, although the pulse pressure is the same in the two cases. *Quite so. The above remark is of exactly* the same order as would be one to the effect that, strangely enough, a mover carried a trunk from the ground floor to the second story without any trouble but gave out in going from

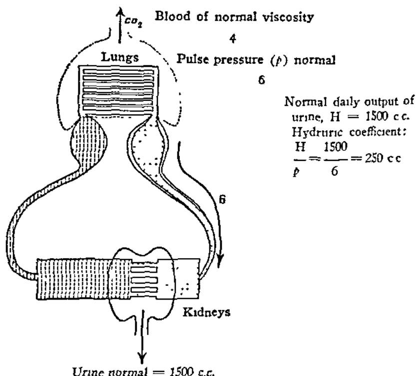



Fig. 755.—Normal subject (pulse pressure in centimeters of mercury).

the sixth to the seventh, yet he was carrying the same trunk and the number of steps to be climbed was the same in both instances. Precisely so; but the man had been exhausted owing to the five stories already climbed. The same applies to the heart, in order to climb the 70 steps from 190 to 260, the heart had already to climb the 190 steps represented by the diastolic pressure and was exhausted; on the other hand, the heart was quite fresh in climbing the 70 steps from 90 to 160, having as yet ascended only 90. Nothing, it would seem, could show more definitely than this the importance of the diastolic pressure.

As a matter of fact—in general clinical work—cases of high arterial pressure may be roughly divided into the four following groups.

1. Plethoric cases.
2. Nervous (angiospastic) cases
3. Renal (infected and poisoned) cases.

The natural ultimate ending in these three groups is represented in the last group.

4. Arteriosclerotic cases.

The first three categories correspond to perfectly distinct clinical conditions, of different pathogenesis, and consequently yield definite therapeutic indications This classification, therefore, serves some practical purpose. Yet, in truth, these several conditions may perfectly well occur in combination: A plethoric subject (*e g*, weighing 100 kilograms) may, in addition, be angiospastic (abnormally emotional) and harbor infection (syphilis) Under these circumstances he is very likely to travel a precipitate journey along the road leading to arteriorenal sclerosis (arteriosclerosis), this constituting the natural penultimate stage in chronic high arterial pressure. A diagram illustrating the course followed by these instances of cardiorenal sclerosis is presented herewith It will be noticed, however, that this next to last, almost incurable stage, that of arteriosclerosis, is preceded by a long period of functional hypertension (plethoric, angiospastic, and renal), in respect of which we are possessed, on the contrary, of very efficient remedial means The differential diagnosis of the several varieties of high arterial tension is thus by no means merely an academic procedure.

### I.—PLETHORA.

The term plethora actually applies to a very clear-cut and frequent clinical state. It is not employed in current text-books on internal medicine because the time-honored list of diseases includes scarcely more than the disorders accompanied by known organic lesions, definite humoral disturbances, or distinct symptom-groups

The plethoric subject, indeed, is by no means an ill person in the accepted sense of the word Apart from slight, occasional

disturbances such as skin eruptions, hemorrhoids, etc., he is in a flourishing state of health and apparently normal, in fact he displays an excess of functional activity betokening unusually intense vitality. He consumes large amounts of food and his digestive functions are admirably performed—as, indeed, is likewise the case in diabetic, gouty, or obese cases. He takes in large quantities of fluid and is polyuric, as are diabetic and gouty

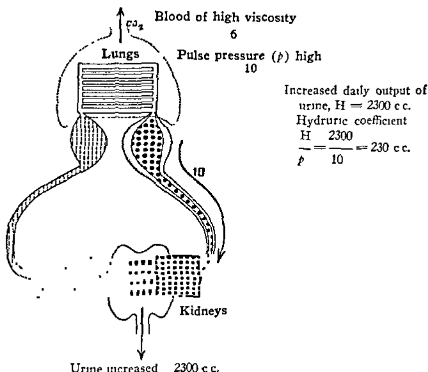



Fig 756—Plethoric subject (pulse pressure in centimeters of mercury).

patients. His complexion is ruddy and his appearance that of robust health. Without his being, properly speaking, an obese individual, his weight is nevertheless distinctly above normal, *e g.*, 96 kilograms (212 pounds) as against a height of 187 centimeters (6 feet 1½ inches); 74 kilograms (163 pounds) as against a height of 166 centimeters (5 feet 5 inches), etc. He has marked endurance, is very active, and the aggregate of work he performs may be much above the normal—as in many gouty and diabetic cases

In short, without being at all ill, one might almost say that the plethoric subject is a supernormal individual, a "superman" from the physiological standpoint. The heart, unusually powerful, hypertrophied, contracts with unusual vigor, this being reflected in a heightened pulse pressure. The blood, richer and less watery, exhibits a higher degree of viscosity. The kidneys, adapted to an unusually abundant circulation and nutritive process, eliminate abnormally large amounts of water, salt, urea, uric acid, etc The digestive glands, richly supplied with blood, are overactive in their secretion, with resulting polyphagia, polydipsia, polyuria, plethora, etc.

The plethoric is thus an individual who is not, properly speaking, abnormal, but supernormal, featured by his appearance of flourishing health, his body weight which exceeds the normal, and his high blood-pressure and viscosity.

He is, however, a candidate for obesity, diabetes, or gout, of which he already presents many anatomical and functional manifestations He is a candidate for sclerotic vasculorenal disorders To the author's mind, it is precisely a great feature of superiority of the sphygmo-visco-hydrurimetric methods that they are sufficiently searching to disclose the morbid tendencies long before any established and recognized pathological change exists, hence enabling the physician to rectify them with much greater certainty.

## II.—NERVOUS ANGIOSPASTIC CASES.

Everything tends to show that this condition frequently constitutes the intermediate stage between simple plethora and cardiorenal sclerosis—that it represents in part what Huchard so appropriately termed the stage of presclerosis

In the course of the development of sclerosis, starting from the simple plethoric subject, the present stage, that of presclerosis, might be said to come 5 years after the onset, and that of established, irremediable sclerosis 10 to 15 years after the onset

At times, indeed, one is enabled to contrast the two clinical types in an almost perfect manner. Thus, the case is recalled of a man of 31 years, obese and suffering from lithiasis, 171 centimeters (5 feet 7 inches) tall and weighing 123 kilograms (271

pounds), florid and well compensated, with a pulse rate of 98, systolic pressure 250, diastolic pressure 120, and viscosity 4.8; and the case of his mother, 51 years of age, of the degenerated obese type, 160 centimeters (5 feet 3 inches) tall and weighing 87 kilograms (192 pounds), an established case of sclerosis, with nycturia, albuminuria, a pulse rate of 120, systolic pressure of 280, diastolic pressure 160, and viscosity 4.1

One of the characteristic features of this condition, whether as regards the pulse rate, the systolic and pulse pressures, the blood viscosity, or the output of urine, is an *altogether abnormal instability* or *variability* which is met with neither in the preceding stage—practically normal from the circulatory standpoint—nor in the subsequent stage in which permanent pathological changes have occurred

In the first stage balance is maintained by virtue of a general, harmonious, and regular functional hypertrophy; in the third stage, the system has adapted itself, either well or badly, to permanent lesions constituting a permanent infirmity, it continues on its course, limping along in a continuous, even manner, as it were

In the stage of the disease here especially under discussion, however, the organism, not yet permanently altered, is not resigned to its fate and will not admit itself conquered It struggles against the approaching collapse by processes of compensatory hypertrophy; but at times the functional adaptation becomes insufficient, there is functional disorganization, disordered reactions, angiospasms, and various forms of insufficiency manifested in the form of paroxysmal attacks of hydremia, angina, *myocardial weakness, etc. Ordinarily, under the mere* influence of a diet instinctively imposed upon himself by the patient, everything returns to normal. But the transitory attacks of hydremia with diminished urinary output, high blood-pressure, and increased blood viscosity, expressed in a sudden, marked rise of the sphygmoviscosimetric index and betokening a forcible reaction on the part of the heart against an abrupt vasculorenal block due to angiospasm, are quite significant and characteristic. They constitute the last cry of warning at the entrance to the blind defile of sclerotic infiltration.

The outstanding feature of this stage is *nervous erethism, emotivity*, and *exaggerated tendency to angiospasm* which, in predisposed subjects, plays an important rôle in the pathogenesis of arteriosclerosis, as Lancereaux, Bouveret, Potain, and Huchard clearly saw and taught. *Some cases may even reach ultimate sclerosis solely by the angiospastic route. The majority reach it, however, by the plethoric route; degeneration is promoted by angiospasm and infections.*

### III—RENAL CASES. NEPHRITIS.

The marked influence of *nephritis* (excepting certain rare forms of excessive renal permeability) in causing high blood-pressure is an obvious clinical fact, whatever explanation of it be adopted. Whether the nephritis be acute or chronic, of infectious or toxic origin, it is nearly always associated with high blood-pressure, which is slight or wanting in simple albuminous nephritis, moderate in chloridemic forms, constant and sometimes very marked in the azotemic and hydremic forms The last-mentioned form, indeed, as is well known, was long termed hypertensive nephritis, high blood-pressure constituting one of the cardinal features of this type of the disease (see *Albuminuria*).

Whether the nephritis be the result of a metabolic disorder such as gout, or of some form of poisoning such as lead poisoning, or of an infectious disease such as scarlet fever or typhoid fever, high blood-pressure is one of its most constant symptomatic manifestations, and frequently, indeed, it exhibits distinct features in accordance with the clinical courses of these several disorders.

A rational classification of these cases is afforded by systematic study of the renal functions (output of water, chlorides, urea, albumin, etc ), in conjunction with blood-pressure determinations

### IV—ARTERIOSCLEROSIS.

Plethora, angiospasm, and infection, whether they do or do not cause preliminary renal changes, and whether they are or are not present in combination, in the long run induce degeneration of the

pounds), florid and well compensated, with a pulse rate of 98, systolic pressure 250, diastolic pressure 120, and viscosity 4.8; and the case of his mother, 51 years of age, of the degenerated obese type, 160 centimeters (5 feet 3 inches) tall and weighing 87 kilograms (192 pounds), an established case of sclerosis, with nycturia, albuminuria, a pulse rate of 120, systolic pressure of 280, diastolic pressure 160, and viscosity 4.1.

One of the characteristic features of this condition, whether as regards the pulse rate, *the systolic and pulse pressures, the blood* viscosity, or the output of urine, is an *altogether abnormal instability* or *variability* which is met with neither in the preceding stage—practically normal from the circulatory standpoint—nor in the subsequent stage in which permanent pathological changes have occurred.

In the first stage balance is maintained by virtue of a general, harmonious, and regular functional hypertrophy; in the third stage, the system has adapted itself, either well or badly, to permanent lesions constituting a permanent infirmity; it continues on its course, limping along in a continuous, even manner, as it were.

In the stage of the disease here especially under discussion, *however, the organism, not yet permanently altered*, is not resigned to its fate and will not admit itself conquered. It struggles against the approaching collapse by processes of compensatory hypertrophy; but at times the functional adaptation becomes insufficient, there is functional disorganization, disordered reactions, angiospasms, and various forms of insufficiency manifested in the form of paroxysmal attacks of hydremia, angina, myocardial weakness, etc. Ordinarily, under the mere influence of a diet instinctively imposed upon himself by the patient, everything returns to normal. But the transitory attacks of hydremia *with diminished urinary output, high* blood-pressure, and increased blood viscosity, expressed in a sudden, marked rise of the sphygmoviscosimetric index and betokening a forcible reaction on the part of the heart against an abrupt vasculorenal block due to angiospasm, are quite significant and characteristic. They constitute the last cry of warning at the entrance to the blind defile of sclerotic infiltration.

One should bear in mind the *frequent occurrence of hemorrhagic complications* such as epistaxis, hemoptysis, and hematemesis.

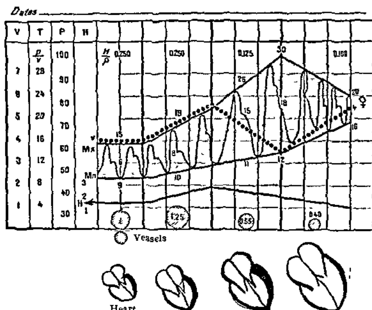



Fig 758—Successive stages in the development of cardiorenal sclerosis

*1* Normal
*2* Eusystoly —Simple plethora —Good cardiorenal compensation.
*3* Hypersphyxia —Hydremia —Renal insufficiency
*4* Heart weakness —Hydremia and anoxemia —Cardiorenal insufficiency
*Mx* Systolic blood-pressure.
*Mn* Diastolic blood-pressure
*V* Blood viscosity.
*H* Daily output of urine
$\frac{H}{p}$ Hydruric coefficient

The main diagnostic features of these disorders will be found in the accompanying table.[1]

[1] In connection with the Note at the foot of the table it should be mentioned that the biometric formula, $\frac{H}{p} < 200$ = sclerosis, is not applicable in aortic insufficiency, the defect in the aortic sigmoid leaflets *completely altering* the dynamics of the cardiac motor, the *efficiency of which* is much reduced on this account

arteries and *arteriorenal sclerosis* or *arteriosclerosis.* In the presence of this common disorder, *continuous elevation of blood-pressure* being the rule, the equation high blood-pressure = arteriosclerosis was long accredited, *though this is manifestly an error,* as already noted.

The practitioner should *make a methodical search for:*

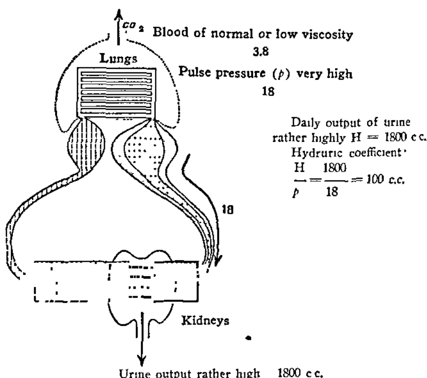



Fig 757.—Subject with sclerotic disease

*Evidences of peripheral fibrosis:* Tortuous temporal vessels, induration of the radial, brachial, and temporal arteries, intermittent claudication, etc

*Evidences of chronic aortitis and cardiac hypertrophy:* Accentuation of the second aortic sound, gallop rhythm, and *enlargement* of the area of heart dulness.

*Evidences of interstitial nephritis:* *Increased output of urine of low specific gravity, low urea output in the urine, high blood-pressure, slight albuminuria, reduction of the hydruric coefficient, and hydremia (lowered blood viscosity).*

| | | | | |
|---|---|---|---|---|
| | | ible only with difficulty | *gravity, nycturia*<br>Albuminuria slight or absent<br>Minor hemorrhages<br>Sometimes gallop rhythm<br>Minor evidences of nephritis, such as headaches, vertigo, dead finger phenomenon, cramps, and epistaxis | Diet low in nitrogen, chloride, or water.<br>Counterirritation, wet cupping |
| Sclerosis.<br>End-result of:<br>Plethora; angiospasm<br>Infections (syphilis)<br>Senile degeneration.<br>Nephritis. | Intoxication, auto- or exo- genous<br>Infections { Malaria, Syphilis, Typhoid fever.<br>Senility | I Stage of compensation.<br>Marked blood-pressure elevation (220 to 300), continuous and partly *irreducible*<br>Lowered blood viscosity.<br>II Stage of lost compensation.<br>Gradually decreasing high blood-pressure, with increasing diastolic pressure<br>Progressively increasing high blood viscosity | I Stage of compensation (cardiorenal symptoms)<br>Evidences of peripheral sclerosis.<br>*Evidences of chronic aortitis*<br>Evidences of interstitial nephritis.<br>II Stage of lost compensation.<br>Symptoms of progressive heart failure and uremia. | *Diuretics and laxatives.*<br>Diet low in nitrogen, chlorides, or water.<br>Monthly wet cupping in the lumbar regions<br>Counterirritation<br>Digitalin. Theobromine. |

## Casual or Induced Elevations of Arterial Pressure.

A. **Casual (accidental).** Following exertion, hiking, running, emotional stress, fear, surprise, a hearty meal, dietary excesses.

B. **Induced.** Artificial tests of the circulation

NOTE.—The term hydruric coefficient is applied by the writer to the ratio $\frac{H}{P}$ of the daily output of urine H to the differential or pulse pressure P, as determined with the Pachon oscillometric sphygmomanometer It equals or exceeds 250 c c in normal subjects and in simple plethora, is sometimes below, at other times above, 250 c c in angiospastic cases, and is always below 200 c c, in cases of sclerosis

## HIGH BLOOD-PRESSURE.

| | Causes. | Special Features. | Main Clinical Evidences. | Relieved by: |
|---|---|---|---|---|
| **Permanently High Arterial Pressure.** | | | | |
| **Plethora.**<br>**Simple plethora.**<br>**Gout. Diabetes.**<br>**Obesity.** | Inheritance of similar state<br>Overeating<br>Sedentary mode of life. | Stable elevation of blood-pressure (180 to 220) *of moderate degree and for a long* time reducible<br>*Proportionate increase* of blood viscosity | Middle-aged subject. 30-50.<br>Appearance of flourishing health.<br>Evidences of active congestion<br>Polyuria, polyphagia, and polydipsia<br>So-called arthritic phenomena (gout, glycosuria, hemorrhoids, etc.) | General reduction of diet<br>Myotherapy.<br>Iodine and iodides<br>Uricolytic agents<br>Purgatives. Mineral spring cures<br>Venesection |
| **Angiospasm.**<br>**Neurovascular erethism.**<br>**Undue emotional excitability.** | Neurotic heredity.<br>Overstrain.<br>Insomnia.<br>Poisoning (lead, nicotine). | Variable elevation of *blood-pressure* (180 to 220) of moderate degree and paroxysmal<br>*Markedly* variable blood viscosity. | Pale and often delicate appearance.<br>Vasomotor phenomena (pallor, flushing)<br>Paroxysmal attacks, pseudoangina, etc.<br>Nervous erethism, neurotic stigmata, exaggerated reflexes | Methodical mode of living<br>Psychotherapy.<br>Hydrotherapy<br>Sedatives to the nervous system<br>Bromides, *valerian, and* borneol ethers<br>Chloral hydrate and *other hypnotics* |
| **Nephritis.**<br>**Acute.** | Acute: infections. | Marked elevation of blood-pressure, reducible *only with* difficulty. | Infectious febrile state, with albuminuria, casts, and hematuria. | Milk diet.<br>Treatment for infections.<br>Counterirritation<br>Calcium chloride. |
| **Chronic.** | Chronic sclerosis | Marked elevation of blood-pressure, *reduc-* | Increased output of *urine of low specific* | |

frequently coupled with a tympanitic note. The patient, in addition to experiencing pain in the hypochondrium and left side of the chest, generally complains of heart disturbances, such as palpitations, dyspnea, and even "missed beats" or extrasystoles, especially when lying on the left side; he states that he is frequently relieved by eructations, not rarely, while he is talking, he may be caught in the very act of swallowing air, conscious or unconscious sialorrhea is an almost constant accompaniment. These patients actually consult

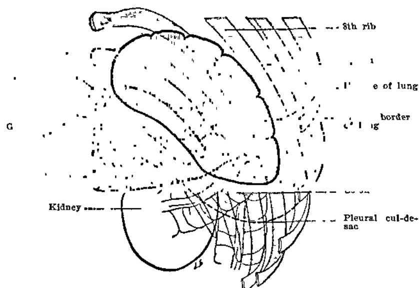



Fig 760—Topographic features of the spleen Projection of the spleen on the costal wall (*Picou, in Poirier and Charpy*)

the physician on account of heart disturbances such as palpitations, precordial pains, "missed beats," angor, dyspnea, etc, much oftener than they do on account of the hypochondriac discomfort, thus justifying the old clinical aphorism "When a patient complains of his heart, there are 9 chances out of 10 of his being a neurotic dyspeptic" Many instances of pseudoangina pectoris arise solely from this cause Gaseous distention of the stomach, having once been detected, its cause remains to be discovered, whether it be a *gastric neurosis, gastric hyperactivity, chronic intestinal disease,* or even *cholelithiasis or appendicitis,* without forgetting the most frequent cause of all, *viz, aerophagia.*

# HYPOCHONDRIUM, LEFT, PAIN IN.

[ὑπό, *below*; χόνδρος, *cartilage*; *below the ribs.*]

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For clinical purposes the left hypochondrium consists, as its etymological derivation shows and without any need of further definition, of that region of the abdomen which is situated beneath the lower and anterior margin of the thoracic cage on the left side Painful affections of the fundus of the stomach, the

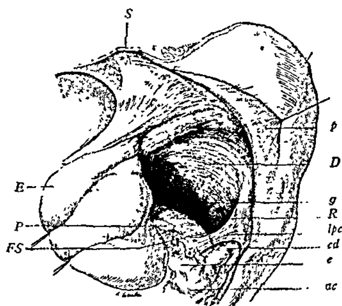



Fig 759—The splenic fossa.

*FS.* Splenic fossa. *E.* Stomach retracted to the right *R.* Upper end of right kidney, with the adrenal gland within *P* Section of tail of pancreas *ac.* Splenic flexure drawn down. *cd.* First part *e.* Omentum of descending colon *lpc* Phrenocolic ligament. *g.* Sulcus formed between the kidney and costal wall *D.* Diaphragm *p.* Thoraco-abdominal wall. *S.* Sternum (*Picou,* after *Constantinesco,* in *Poirier and Charpy*)

spleen, the left kidney, and the splenic flexure of the colon are the chief disorders which find clinical expression in this region.

**Clinical Description.**—Nine-tenths of all instances of pain in the left hypochondriac region, coincidently with pains in the lower (infracardiac) thoracic region on the left side, are of gastric origin and are symptomatic of gaseous distention of the fundus of the stomach, *i e.,* of *flatulence, meteorism,* and *aërophagia.* Percussion leads to the discovery of a pronounced extension of Traube's space,

to the diagnosis. Detection of perinephric abscess frequently demands considerable clinical sagacity, and the author cannot escape the recollection of a disastrous case of perinephric abscess coming on insidiously in a pregnant woman and gradually manifested in persistent pain in the left hypochondrium and lower intercostal spaces, with fever and tenderness in the left lumbar

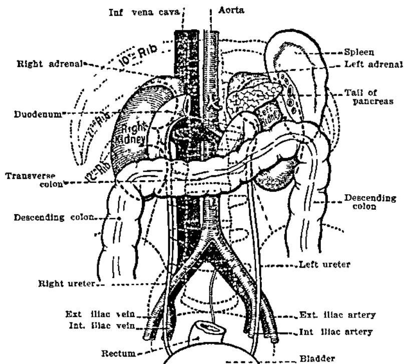



Fig 762—Deep-lying structures in the right and left hypochondria

region, then in an alarming symptom-group of peritonitis, without renal evidences being apparent at the time, and which, although suspected to be of renal or perirenal origin by the first three physicians called, was wholly overlooked by two eminent consultants, who diagnosticated appendicitis and subsequently *failed to be enlightened, after a fruitless appendectomy,* by a pyemic state of daily increasing gravity and even a copious discharge of pus from the urinary tract This was one of the most trying episodes of the author's entire medical career, and one which he is unable to recall without a feeling of bitterness

**Frequency.**—In the order of frequency, surgical affections of the left kidney—nephrolithiasis, pyo- and hydro- nephrosis, perinephric abscess, tuberculosis of the kidney, or neoplasm—come next to the gastric disturbances. Certain details concerning the pain of renal involvement are presented in the section on the

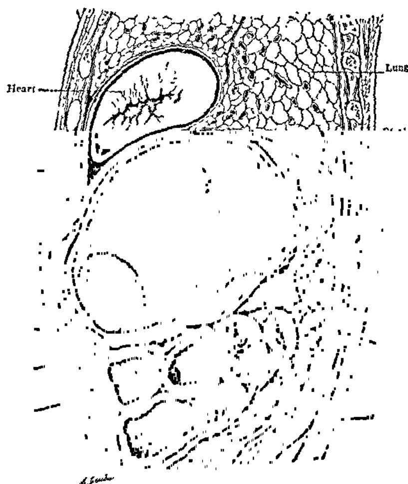



*Fig 761.—Sagittal section through the left hypochondrium, passing in the middle of the space between the parasternal and nipple lines, in a subject with marked dilatation of the stomach (Luschka).*

right hypochondrium *(q. v.)*. Pain on pressure is usually most marked in the lumbar region, it radiates along the left ureter, over the iliac fossa, and even to the testicle. Bimanual palpation may sometimes reveal the presence of a tumor of the kidney; finally, examination of the urine in some instances leads directly

noted and left-sided pleurisy is almost constantly present; frequently, beneath the false ribs of the left side, there is an actual cake-like condition about the spleen.

2. Less commonly, there occur abscesses of the left inter-hepato-diaphragmatic fossa, bounded on the right by the suspensory ligament, posteriorly by the triangular ligament, below by the upper surface of the left lobe of the liver and part of the anterior aspect of the stomach, above and to the left by the diaphragm, anteriorly by adventitious adhesions between the diaphragm and the anterior root of the left lobe and by a varying portion of the anterior abdominal wall.

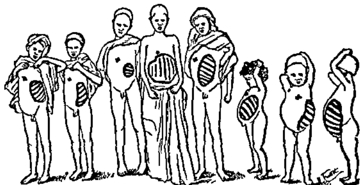


Fig 763—Arab children of Algeria with malarial enlargement of the spleen The cross + shows the position of the umbilicus (*Brumpt*)

The cause of this type of abscess is invariably a perforation of the anterior wall of the stomach

In this variety the initial pain is predominantly situated on the left and sometimes radiates toward the left shoulder, convexity is especially pronounced on the left; Traube's space is always modified; frequently there are signs of left-sided pleurisy, displacement of the heart-apex upward and inward, formation of a mass below the ribs on the left side, and immobilization of the left side of the chest

The splenic flexure of the colon, forming, as is well known, an extremely acute angle, is one of the "critical" points of the intestine, and is one of the commonest sites of intestinal tumors. Indeed, a neoplasm of the splenic flexure frequently causes no local pain, careful deep palpation of the left hypochondrium

Pride, conceit, and absurd pretentions to a species of infallibility may lead physicians to really criminal acts.

Although relatively common, the disorders most specifically attaching to the left hypochondrium are the affections of the spleen. All splenic enlargements, whether due to leukemia, anemia, malaria, syphilis, or polycythemia, and whether or not associated with hepatic cirrhosis or heart weakness, are accompanied by a varying degree of painful tension in the left hypochondrium. Enlargement of the spleen having been noted, there remains to be made, by means of the customary methods of examination (history, blood examination, liver examination, uranalysis, Wassermann reaction, etc ), a diagnosis of leukemia, malaria, syphilis, cirrhosis, heart failure, etc.

**Subdiaphragmatic abscess on the left side** appears to warrant a somewhat extensive consideration in view of the fact that little attention is generally paid to it and because, although uncommon, it is much less rare than one might suppose, the author having personally observed a dozen cases, aside from others that may have been overlooked [1]

There are *two anatomic varieties of subdiaphragmatic abscess* which may give rise to signs and, in particular, to pain in the left hypochondrium These are:

1. More often, **abscesses of the perisplenic fossa** (or *gastro-splenic fossa* of Dieulafoy), bounded above by the diaphragm and the left extremity of the left lobe of the liver, within by the fundus of the stomach and the pancreas, posteriorly by the diaphragm and kidney, anteriorly by the diaphragm and the omentum, externally by the diaphragm and ribs, and below by the splenic flexure and the left mesocolic fold.

Abscesses collecting in this locality are secondary either to *abscesses of the spleen* (*infarct, malaria*) or to a *perforation of the stomach* situated near the cardia or on the posterior wall of the fundus.

*In these cases the pain is located deeply in the left hypochondrium*, the mass formed shows little tendency to encroach on the epigastrium, pronounced enlargement of splenic dulness is

[1] See also Martinet "Des variétés anatomiques d'abcès sous-phréniques," Paris, 1898.

noted and left-sided pleurisy is almost constantly present; frequently, beneath the false ribs of the left side, there is an actual cake-like condition about the spleen.

2. Less commonly, there occur **abscesses of the left inter-hepato-diaphragmatic fossa**, bounded on the right by the suspensory ligament, posteriorly by the triangular ligament, below by the upper surface of the left lobe of the liver and part of the anterior aspect of the stomach, above and to the left by the diaphragm, anteriorly by adventitious adhesions between the diaphragm and the anterior root of the left lobe and by a varying portion of the anterior abdominal wall.

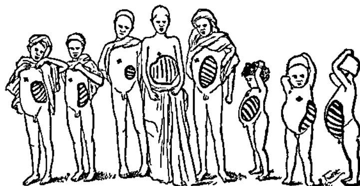


Fig 763—Arab children of Algeria with malarial enlargement of the spleen The cross + shows the position of the umbilicus (*Brumpt*)

The cause of this type of abscess is invariably a perforation of the anterior wall of the stomach.

In this variety the initial pain is predominantly situated on the left and sometimes radiates toward the left shoulder; convexity is especially pronounced on the left; Traube's space is always modified; frequently there are signs of left-sided pleurisy, displacement of the heart-apex upward and inward, formation of a mass below the ribs on the left side, and immobilization of the left side of the chest

The splenic flexure of the colon, forming, as is well known, an extremely acute angle, is one of the "critical" points of the intestine, and is one of the commonest sites of intestinal tumors Indeed, a neoplasm of the splenic flexure frequently causes no local pain; careful deep palpation of the left hypochondrium

may, however, yield significant information which, when correlated with a more or less pronounced symptom-group of obstruction, may lead to the diagnosis of tumor, to be *subsequently con-*

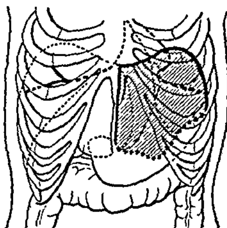


Fig 764—Left-sided abscess between the liver and diaphragm. Perforated stomach, due to gastric ulcer.

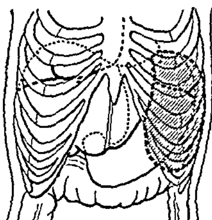


Fig 765—Perisplenic abscess. Abscess of the spleen Infarction, malaria, gastric perforation

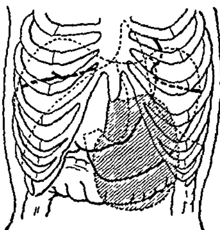


Fig. 766.—Post-gastric abscess.

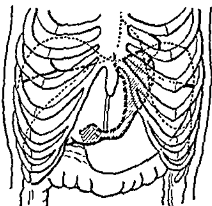


Fig. 767.—Abscess between the liver and stomach.

*firmed* by testing of the stools for blood and fluoroscopy following a bismuth meal.

There is one symptom-group, however, which has not yet been adequately described: *Chronic pain in the left hypochondrium with periodic exacerbations, apparently the result of gaseous accumulation at the splenic flexure and of defective*

transit through the large intestine owing to excessive angulation of the splenic colon—a condition admirably demonstrated by fluoroscopic examination.

The possible occurrence of pain in the left hypochondrium in cases of *pneumonia involving the left lobe* or of *pleurisy* lends itself to the same observations as are made in the section on the right hypochondrium (*q v*).

* * *

As a suggestive instance of the disturbances—sometimes apparently very serious—that may be brought on by the accumulation of air and gases in the fundus of the stomach, with the resulting pressure on the left side of the diaphragm and displacement and distortion of the heart and aorta, the following data pertaining to a case of this type may be here presented:

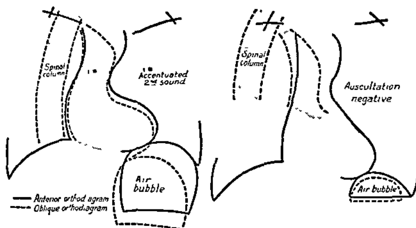



Fig 768.

Case 3596

Man, 1862, $\frac{171 \text{ cm}}{27 \text{ cm.}}$; 76 kilograms

Pressures $\frac{230}{125}$ Pulse press, 100

Marked anginoid syndrome, especially pronounced on walking and after meals During attacks, the patient is "rooted to the spot" and unable to move. The stomach shows a very large air bubble and there is meteorism with marked pressure displacement of the heart and distortion of the aorta

Case 3596

Same patient three weeks later, after treatment Weight, 74.8 kilograms

Pressures $\frac{160}{105}$ Pulse press, 60

All anginoid manifestations have disappeared Locomotion and moderate outdoor sports resumed Meteorism has disappeared and the gastric air bubble is markedly reduced. The heart and aorta are restored to their normal shape and position.

## PAIN IN THE LEFT HYPOCHONDRIUM.

| Type of Pain. | Findings on Palpation and Percussion | Fever Blood Examination | Urinary Manifestations | History and Clinical Course |
|---|---|---|---|---|
| **Flatulence** (aërophagia, dyspepsia, neuroses, and chronic gastrointestinal affections) | | | | |
| Feeling of tension and puffiness rather than actual pain.<br>Recrudescence after meals, as a rule | Meteorism, unusual tympanitic resonance, enlargement of Traube's space<br>Undue tension of the local tissues | 0 | 0 | Neurosis.<br>Mild. |
| **Surgical affections of the left kidney** (nephrolithiasis, pyo- and hydro- nephrosis, perirenal abscess, tuberculosis of the kidney, or neoplasm). | | | | |
| Variable according to the cause. Paroxysmal (renal colic) in nephrolithiasis<br>Constant in perirenal abscess<br>Frequently wanting in tuberculosis. | Tenderness on palpation, especially in the lumbar region<br>Kidney sometimes discernible on bimanual palpation and manifestly enlarged | + with leucocytosis in infections. | Excessively acid reaction, Excessive uric content, Gravel } In nephrolithiasis.<br>Pyuria in infections.<br>Hematuria in neoplasm | Neuroarthritic diathesis or infection |

**PAIN IN THE LEFT HYPOCHONDRIUM** (*continued*).

| Type of Pain | Findings on Palpation and Percussion | Fever Blood Examination | Urinary Manifestations | History and Clinical Course |
|---|---|---|---|---|
| **Affections of the spleen, splenic enlargement** (malaria, syphilis, leukemia, polycythemia, heart failure, or infarction) | | | | |
| Feeling of tension rather than pain. | Enlargement of spleen, sometimes noticeable on palpation. | 0 in heart failure<br>+ in malaria or leukemia<br>Variable in syphilis<br>W + in syphilis | Variable<br>Sometimes albuminuria | Malaria<br>*Syphilis*<br>Heart disease. |
| **Subdiaphragmatic abscess** | | | | |
| Variable | See annexed illustrations | + with leucocytosis | Occasionally, "urinary vomica" | Often obscure<br>Always serious. |
| **Tumor of the splenic flexure.** | | | | |
| *Frequently wanting.* | Sometimes tenderness below the spleen and an appreciable bulging | 0 | 0 | ?<br>Advanced age, progressive obstruction, cachexia |

# HYPOCHONDRIUM, RIGHT, PAIN IN.

---

The right hypochondrium normally corresponds to the lower border and anterior surface of the liver in its entire extent. As a matter of fact, *four-fifths of all pain disturbances in this region originate in the liver or gall-bladder;* yet by no means all pains in the hypochondrium originate in these structures.

* * *

A brief review of the regional anatomy will suggest the various possibilities in the interpretation of pain in this area.

The right hypochondrium is in its entirety in relationship with the *anterior surface* and *lower border of the liver,* including, in its middle portion, the fundus of the gall-bladder. *Disorders of the liver and other biliary structures* constitute the chief pain-producing agencies in the right hypochondriac region. Acute, paroxysmal pains of the type of colic are especially characteristic of *cholelithiasis,* simple or with complications It should not be overlooked, however, that frequently this disorder is—like chronic appendicitis—spontaneously manifested only in what appear to be ordinary dyspeptic disturbances or in a pyloric symptom-group with tendency to stasis and dilatation in the presence of adhesions uniting the gall-bladder and duodenum Jaundice occurs in about one-fourth of all cases of cholelithiasis. Only in exceptional instances (1 out of 20) will palpation not induce a characteristic pain at the fundus of the gall-bladder for a more or less prolonged period of time during the intervals between the acute attacks

Pain of hepatic origin is generally of a duller type, and is sometimes only brought out by palpation or percussion. Pain has to be actually examined for in these cases Sometimes it is latent; *active or passive congestion* of the liver is by far the commonest of the disorders of this organ inducing sensitiveness; it accompanies, precedes, or heralds the oncoming of the majority of instances of

hepatic cirrhosis. As is well known, it is one of the most constant symptoms of *heart weakness* or *heart failure.*

Lastly, one should always bear in mind the possibility of *syphilis of the liver,* a fairly common but generally rather painless condition (enlarged syphilitic liver, pseudocancerous form, syphilitic

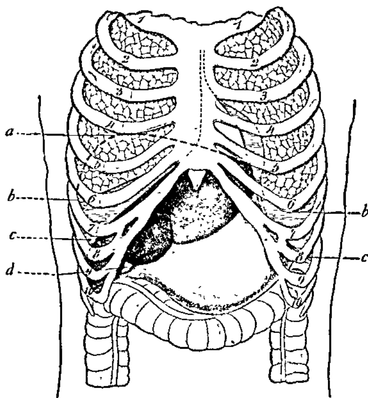



Fig 769.—Anatomic relations of the liver with the walls of the chest and abdomen (*Laederich*) *a* Projected outline of the upper border of the liver *b* Lower border of the lung *c* Pleural cul-de-sac. *d* Gall-bladder

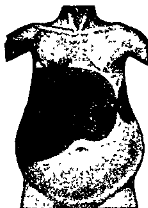
lobulated liver, lobed liver, or syphilitic cirrhosis); of *hepatic abscess* (history of malaria, fever, jaundice, pain, etc ), and of *cancer,* fortunately much more uncommon ("galloping" enlargement of the liver with marked emaciation and rapid cachexia). In all these cases, it is not the character of the pain but the associated signs and symptoms and the clinical course which constitute the basis of the

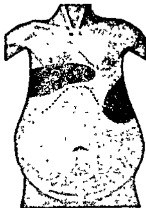
diagnostic conclusion—as will be seen in the tabular summary at the end of this section.

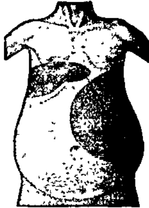
Immediately behind the liver are situated the angle formed by the *ascending and the transverse colon* (hepatic flexure) and the *first flexure of the duodenum*, to which, furthermore, the gall-bladder so frequently becomes adherent in the event of pericholecystitis. A corollary of this is that pain in the right hypochondrium may be symptomatic of a *precolic or retrocolic high appendicitis*, and the incipient jaundice frequently present in these cases may still further augment the difficulty of diagnosis. Indeed, the author has seen the two following mistakes committed, *viz.*, a cholecystitis with prolapse mistaken for an appendicitis and a high appendicitis mistaken for cholecystitis. Appendicitis may, as a matter of fact, be the starting-point of subhepatic abscess. Ordinarily, and apart from the particularly acute emergencies in which incipient or established peritonitis prevents all precise localization, palpation will enable the practitioner to elicit an especial localization of the disturbance in the right iliac fossa.

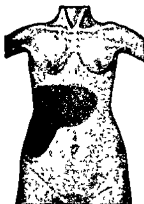
As for *duodenal ulcer* (see *Dyspepsia*) the particular type of the pain, coming on spontaneously and periodically with a uniform relationship to meals, the symptom-group of hyperchlorhydric indigestion which usually accompanies it, and the lower position of the tenderness elicited by palpation between the right hypochondrium and the umbilicus will frequently permit of an easy diagnosis, sometimes definitely confirmed by the advent of hematemesis. The chief condition to be differentiated from duodenal ulcer is cholelithiasis.

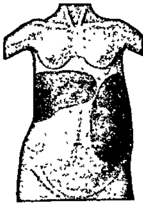
Behind the lower surface of the liver, the hepatic flexure and the duodenum is the *right kidney*, many disorders of which may be manifested in pain in the right hypochondrium. Especial mention should be made of *nephrolithiasis* and of *kidney suppurations* (pyonephrosis) or *perirenal abscess*. Aside from the paroxysmal attacks of *renal colic*, in the presence of which brief hesitation may occur in the differentiation from hepatic colic by reason of the practical impossibility of making an examination on account of the severe pain, *cholelithiasis* is readily differentiated by the fact that the pain or tenderness is most marked in the lumbar region, by the radiation of the pain along the ureters and to the testicles, by the urinary

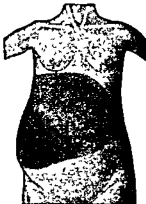

Fig 770 —Alcoholic hypertrophic cirrhosis with ascites

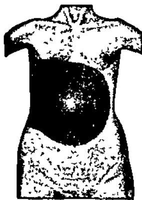

Fig 771 —Alcoholic atrophic cirrhosis with ascites

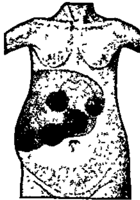

Fig 772 —Banti's disease Greatly enlarged spleen, atrophic cirrhosis, and ascites

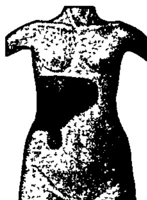

Fig 773 —Hepatoptosis Movable lobe


Fig 774 —Biliary cirrhosis with greatly enlarged spleen


Fig 775 —Biliary cirrhosis with enlarged liver and spleen


Fig 776 —Hydatid cyst of the liver (left lobe)


Fig 777 —Nodular cancer of the liver


Fig 778 —Cancer of head of pancreas Dilated gallbladder

diagnostic conclusion—as will be seen in the tabular summary at the end of this section.

Immediately behind the liver are situated the angle formed by the *ascending and the transverse colon* (hepatic flexure) and the *first flexure of the duodenum*, to which, furthermore, the gall-bladder so frequently becomes adherent in the event of pericholecystitis A corollary of this is that pain in the right hypochondrium may be symptomatic of a *precolic or retrocolic high appendicitis*, and the incipient jaundice frequently present in these cases may still further augment the difficulty of diagnosis. Indeed, the author has seen the two following mistakes committed, *viz.*, a cholecystitis with prolapse mistaken for an appendicitis and a high appendicitis mistaken for cholecystitis. Appendicitis may, as a matter of fact, be the starting-point of subhepatic abscess. Ordinarily, and apart from the particularly acute emergencies in which incipient or established peritonitis prevents all precise localization, palpation will enable the practitioner to elicit an especial localization of the disturbance in the right iliac fossa.

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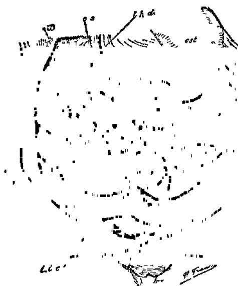


Fig 780—Kink at the first flexure of the duodenum due to a nephroptosis of the third degree Pronounced dilatation of the first part of the duodenum Hydronephrosis has been produced owing to multiple kinks of the ureter. The patient had been admitted to Prof Terrier's service with symptoms suggesting intestinal obstruction She died in a few days without having been operated, and at the autopsy the only lesions found were those here illustrated *I p d*, first portion of the duodenum, greatly dilated above its flexure, *c*. Note the adhesions *ad* *c d*

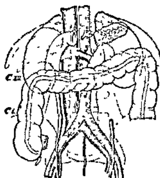


Fig 781.—Diagram showing the normal relations of the right kidney with the hepatic flexure and duodenum The lower pole of the kidney is generally situated behind the hepatic flexure. Exceptionally, this flexure lies below the kidney (*Alglave*).

manifestations, and sometimes by the detection of gravel in the urine. Similar considerations apply in the case of *pyonephrosis* or *perirenal abscess,* although in the latter instance, if the abscess points—as is seldom the case—below the liver, the diagnosis may be very difficult; differentiation is, however, of capital importance, since the institution of proper operative treatment, *viz*, lumbar in-

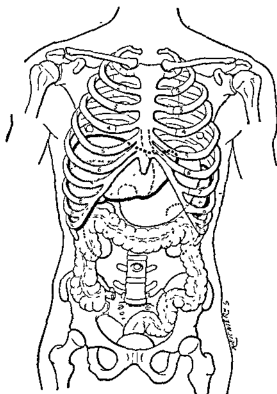


Fig 779—Relations of the abdominal organs, viewed anteriorly.

cision or subhepatic celiotomy, depends upon the decision reached To be particularly borne in mind, in order that they may be excluded, if possible, by careful clinical study, are pericholecystitis, subhepatic abscess of appendicular origin, and subdiaphragmatic abscess.

All the above mentioned disorders, capable of causing pain in the right hypochondrium as an important or predominant clinical manifestation are of abdominal origin, being hepaticobiliary, colo-duodenal, or renal. Two thoracic disorders, *right-sided pleurisy* and *right-sided pneumonia,* may also give rise to pain in this locality.

subdiaphragmatic abscess In the course of a study of the several varieties of subdiaphragmatic abscess, made by the

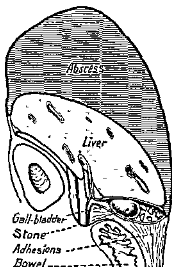



Fig 783—Verticotransverse diagrammatic section passing through the gall-bladder The fundus of the latter exhibits a perforation Below, adhesions are shutting off the peritoneal cavity An outpouring of bile has occurred and the resulting abscess developed between the diaphragm and the liver, which is pushed downward (Right-sided inter-hepato-diaphragmatic abscess).

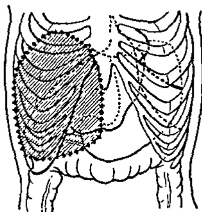


Fig. 784—Right-sided abscess between the diaphragm and liver.

author a number of years ago, this variety was found to have existed in 60 out of 146 cases. Such abscesses are sometimes

In *pneumonia*, such a location of the pain is the rule in involvement of the lower lobe, and at times of the middle lobe of the right lung, particularly in children. In these cases, indeed, pain in the right hypochondrium is actually of threefold origin:

Cutaneous, or pain attending a superficial hyperesthesia (Head's hyperesthetic projection zone).

Hepatic, or a deep seated pain due to the active congestion of the liver attending infection.

Diaphragmatic, or pain due to an inflammatory pleural reaction

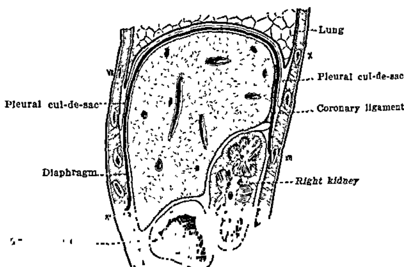



Fig. 782—Anteroposterior section of the liver (*Charpy*). The section passes through the right hypochondrium

Excepting in children, the diagnosis is very seldom held in suspense for a long time in pneumonia. The same is true of *pleurisy*, except in diaphragmatic pleurisy, in which the diagnosis is often difficult.

Lastly, mention should be made of a variety of *subdiaphragmatic abscess* located in the right hypochondrium, in the right interhepatodiaphragmatic fossa, and bounded above by the diaphragm, to the left by the falciform ligament and gastrohepatic omentum, and below by the right half of the transverse colon and a double transverse fold of peritoneum extending from the lower portion of the ascending colon to the abdominal wall slightly below the tip of the eleventh rib. This is one of the commonest varieties of

secondary to a disturbance of the liver, such as cholelithiasis or abscess or suppurative hydatid cystic disease of the right lobe, or may follow perforation of a duodenal ulcer.

The diagnosis is based on the initial pain, most marked in the right hypochondrium and frequently radiating toward the

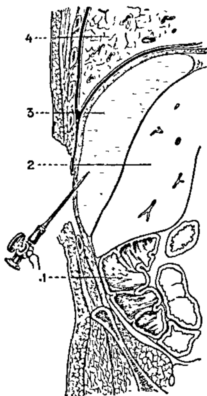
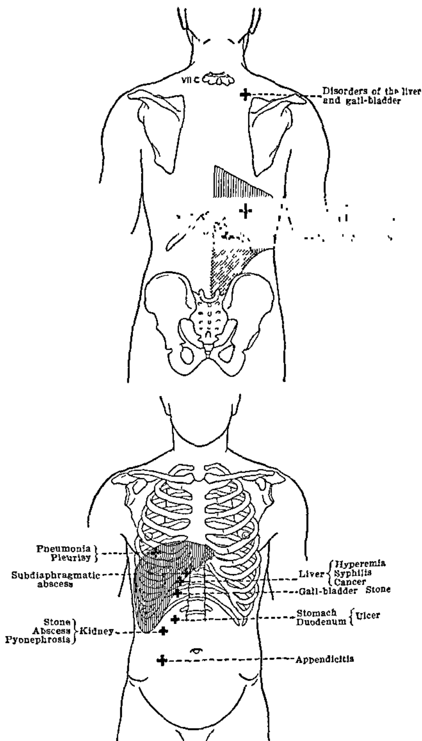



Fig. 787—Evacuation of a subdiaphragmatic abscess (3) of appendiceal origin through the chest wall below the pleura 1 Colon 2 Liver, displaced downward and inward. 4 Right lung (*Kelly*)

right shoulder; on the epigastric prominence, extending mainly toward the right side; on the enlarged area of hepatic dulness, or, on the other hand, in the case of gaseous abscesses, on the percussion note over the liver and the lowered position of this organ, the complete or incomplete immobilization of the right half of the thorax, and the frequent association with a right-sided pleurisy or even a phrenic neuralgia on that side. The




Figs 785 and 786—Points of tenderness in the right hypochondrium, with their respective causes and modes of radiation.

(*c*) *Hydatid cyst* of the liver is usually painless, even where it reaches a considerable size. In *exceptional instances* it may, however, give rise to *attacks of pain suggesting gall-stones.* The cause of these attacks is by no means clear; possibly a reflex contraction

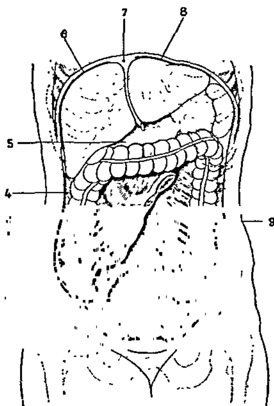



Fig 789—Cavities that may be occupied by pus in peritonitis *1* Inframesenteric space *2* Pelvic cavity *3* Supramesenteric space. *4* Right paracolic space *5* Interhepatocolic space *6*. Right and *8*, left subdiaphragmatic spaces, the latter separated from the preceding by the falciform ligament (*7*) *9* Left paracolic space.

of the biliary passages occurs, constituting a species of painful biliospasm (Chauffard); or, more probably, there is obstruction of the common bile duct by vesicles or membranes set free from an hydatid cystic disease of the liver that has opened into the bile passages (Dèné).

causal diagnosis should be based *mainly on the history* of the case.

Finally, three rarer conditions may be referred to:

(*a*) An exceptional localization of *herpes zoster,* revealed by

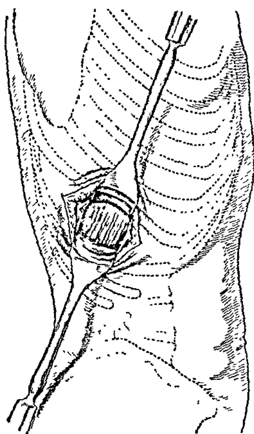


Fig 788—Incision of a subdiaphragmatic abscess of appendiceal origin in the 9th right costal interspace (*Bérard*).

direct inspection of the painful area—a procedure indicated in all regions of the body.

(*b*) A special localization of the "girdle pain" of *tabetic origin,* mere recollection of the possibility of which is sufficient, but which should always be thought of in the presence of pain in the right or left hypochondrium or the epigastrium, and which systematic examination of the reflexes and station—*always a necessary procedure*—is sufficient to establish.

ages of sixty and sixty-five and 5 times oftener in women than in men). The pathogenetic rôle of cholelithiasis in cancer of the gall-bladder is supported by the fact that the latter seems to be getting more uncommon since operations for cholelithiasis have been performed oftener and earlier in the disease. Thus, between 1890 and 1900, 140 operations for gall-stones revealed 17 instances of cancer of the gall-bladder (12 per cent); from 1901 to 1910, 197 operations brought to light 16 cancers (8 per cent.), and from 1911 to 1919, 151 cases yielded only 9 cancers (6 per cent.).

Diagnosis of hydatid cyst simulating gall-stones (hydatid hepatic colic) is a matter *of great difficulty* and *sometimes* quite impossible where the clinical signs of cyst are wanting.

* * *

Courvoisier, in 1890, in his monograph on "The Pathology and Surgery of the Biliary Passages," enunciated the following law: "*In obstruction of the common bile duct by a stone, dilatation of the gall-bladder is exceptional; under these circumstances the gall-bladder is usually contracted. In obstruction of the duct due to any other cause, dilatation of the gall-bladder is the rule.*" This law was confirmed but slightly modified by Terrier, who wrote: "*In cases of occlusion of the common bile duct of internal causation, the gall-bladder is ordinarily shrunken, whereas dilatation of the gall-bladder is the rule when occlusion of the biliary passages is of external causation.*"

Krahenbuhl, of Bâle, in turn, corrects the law in the following manner: "*In cases of gall-bladder enlargement of long standing, the occlusion of the common bile duct is ordinarily of neoplastic nature; where gall-bladder enlargement is wanting or but temporary, occlusion of the duct is usually due to stone.*"

Statistical records confirm the FREQUENCY OF STONES IN CANCER OF THE GALL-BLADDER. Out of 41 cases of cancer of the gall-bladder the presence of stones was found mentioned in 36 instances, or 89 per cent ; in 4 other cases, stones were not referred to, and in a fifth case, the patient is said to have previously passed calculi with his stools.

The relatively frequent coexistence of lithiasis and cancer of the gall-bladder naturally suggests, as J. Dumont states, the thought of *a possible etiologic relationship between the two diseases.* Is cholelithiasis the cause of cancer or merely a consequence of it? A fact justifying the belief that cholelithiasis precedes the cancer and constitutes one of its predisposing causes (irritation theory) is that in the overwhelming majority of cases (37 out of 41), the patients had been suffering for a long time, in some instances for decades, from hepatic colic. (It may be added, to complement the above data relating to cancer of the gall-bladder, that jaundice was present in 63 per cent. of the cases and that cancer occurred oftenest between the

| | | | | | |
|---|---|---|---|---|---|
| **Syphilitic liver.** | Little or no tenderness | Liver smooth and *slightly enlarged* | Rare and slight. | Fever rare. | Specific history. Positive Wassermann |
| **Cancer of liver.** | Inconstant, seldom sharp. | Liver generally enlarged and irregular (nodular in secondary cancer). Sometimes enlarged and smooth (primary cancer). | Frequent; of varying grade | Fever occasional. | "Galloping" loss of weight. Rapid cachexia. |
| | | **Kidneys.** | | | |
| **Nephrolithiasis.** | Paroxysmal attacks of *renal colic* In the intervals· Right kidney or ureter sensitive to palpation, blows, or jarring. | Generally negative | Wanting. | No fever | Neuroarthritic history (plethora, gout, or diabetes) |
| **Perinephric abscess. Hydro- or pyo-*nephrosis.*** | Deep-seated but seldom severe pain. | Tenderness, especially in lumbar region. | Exceptional. | Pronounced fever Leucocytosis. | *Sometimes pyuria or* sudden discharge of urine *Urinary manifestations* |
| | | ***Digestive Tract.*** | | | |
| **Gastric or duodenal ulcer.** | Spontaneous pain at definite intervals after meals. | Greatest tenderness frequently midway between right hypochondrium and umbilicus | Exceptional, except in the event of malignant change and obstruction of bile duct | No fever No leucocytosis. | Symptoms of dyspepsia with hyperchlorhydria, sometimes hematemesis. |

## PAIN IN THE RIGHT HYPOCHONDRIUM.

| Causes. | Kind of Pain. | Findings on Palpation and Percussion | Jaundice | Fever and Leucocytosis | History and Clinical Course |
|---|---|---|---|---|---|
| **Liver.** | | | | | |
| **Simple congestion of liver (active or passive).** | Little or no actual pain; never acute, rather dull. *Elicited* especially on palpation and percussion. | Liver smooth, moderately enlarged, and tender. | Uncommon and slight (subicterus). | Fever only occasionally. | Digestive disturbances, indigestion, alcoholism, *appendicitis*, or circulatory disorders (cardiopulmonary affections) |
| **Uncomplicated cholelithiasis.** | Paroxysmal attacks of hepatic colic. In the intervals: Gall-bladder sensitive to palpation, pain induced by blows or jarring (as in a carriage), radiates to the right scapula. | Gall-bladder **sometimes** palpable and tender. | *Obstructive* jaundice **frequent**, regularly present in the event of impaction of a stone in the bile-duct. Absent in gall-bladder colic. | Only at the time of the attacks; no leucocytosis | Neuroarthritic history (plethora, gravel, gout, etc.), history of infection (typhoid fever) or negative history |
| **Cholelithiasis with acute cholecystitis.** | Sharp and lasting. | Right hypochondrium "board-like," owing to spasm and rigidity of the recti. | **Often slight.** | Fever sometimes of intermittent type, with chills. **Leucocytosis.** | Do. |
| **Abscess of liver.** | Variable; sometimes sharp | Liver more or less enlarged and tender. | **Nearly constant.** | Do. | History of malaria or cholelithiasis. Striking results from emetine, quinine, and incision |

## ILIAC FOSSA, LEFT, PAIN IN.

[Ilia, *the flanks; appertaining to the flanks.*]

---

Practically the same considerations apply to pains in the left iliac fossa as to those in the right iliac fossa (to be discussed in

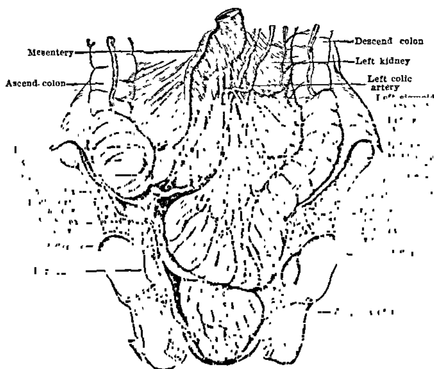



Fig 790—Normal position and relations of the sigmoid flexure and rectum in the adult. The pelvis has been freely opened anteriorly by a frontal section passing through the centers of the acetabular fossæ (*Poirier*)

the succeeding section), the only important differences being those attendant upon the presence of the appendix and cecum on the right side in lieu of the iliac colon on the left.

## PAIN IN THE RIGHT HYPOCHONDRIUM (*continued*).

| CAUSES | KIND OF PAIN | FINDINGS ON PALPATION AND PERCUSSION. | JAUNDICE | FEVER AND LEUCOCYTOSIS | HISTORY AND CLINICAL COURSE. |
|---|---|---|---|---|---|
| Appendicitis (high). | Constant and rather sharp | Tenderness from hypochondrium to right iliac fossa | Rather frequent. | Fever and leucocytosis. | Frequently history of dyspeptic symptoms, such as vomiting, constipation, etc |
| **Lungs and Pleuræ.** | | | | | |
| Pleurisy. | At the onset, same features as intercostal neuralgia | *Friction rub* or *flatness* on right side of chest Liver displaced downward in extensive effusion | Wanting. | Moderate fever | Symptoms of pleurisy, such as cough and dyspnea. |
| Pneumonia. | Occurring coincidently with chill and abrupt rise of temperature | Liver *sometimes* enlarged and tender | Sometimes incipient jaundice. | High fever Leucopenia. | Typical temperature curve Characteristic sputum. |
| **Subdiaphragmatic Abscess.** | | | | | |
| Appendicitis, hepatic abscess, malaria, etc. | Generally a dull, deep-seated pain | Markedly variable according to location of disturbance | Occasional. | High fever. Leucocytosis. | Pus discharge and pus tracts. |

finger. This condition, when correlated with alternating diarrhea and constipation and the discharge of mucus and false membrane, generally affords an easy diagnosis. Pain is not constant in these cases.

It should be borne in mind that *tumors of the descending colon and sigmoid* may be particularly silent from the symptomatic standpoint and be associated with very little local or general disturbance; even the degenerative process itself, similar to a small ring of metal, is often hard to detect. In general, in any subject over fifty years of age exhibiting irregularity of bowel action which had previously been regular, or losing weight, the stools should be examined for blood and x-ray studies carried out.

The annexed anatomical illustration of the region and the diagnostic tabulation will give data serviceable in the clinical study of this region.

Abdominal palpation, palpation from the rectum (and the vagina in women), fluoroscopic examination after a bismuth meal, and gross and microscopic examination of the stools are

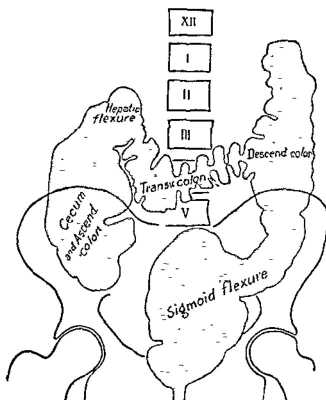



Fig 791—Radiographic view of the large intestine (*Tuffier, Aubourg*)

*the main clinical procedures employed in investigating the left* iliac fossa.

*Muco-membranous enterocolitis* (or mucous colitis), a very common disorder, gives rise to pain localized in the left iliac fossa; it is oftenest situated in the cecum and transverse colon. Again, palpation of the left iliac fossa frequently reveals the descending colon and sigmoid in a contracted condition, with reduced size, imparting the sensation of an elastic "sausage" rolling beneath the

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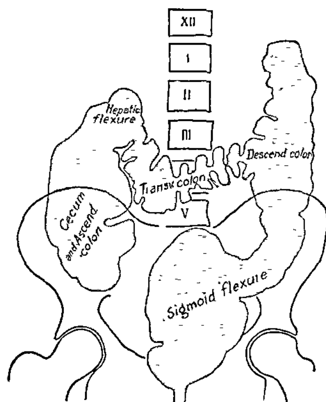



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# ILIAC FOSSA, RIGHT, PAIN IN.

The semeiology of the right iliac fossa revolves chiefly about the diagnosis of acute and chronic appendicitis.

Yet, appendicitis is not the only pain-producing disorder met with in this region.

It would seem of interest in this connection to reproduce the highly suggestive statistics recorded by Cabot in *"Differential Diagnosis."*

*Relative frequency of the disorders causing pain in the right iliac fossa (Cabot, 1747 cases).*

| | | |
|---|---|---|
| Appendicitis | 1169 cases | 66½ per cent. |
| Pus-tube (and pelvic adhesions) | 427 " | 24 " " |
| Dysmenorrhea | 81 " | 4½ " " |
| Extra-uterine pregnancy | 23 " | 1½ " " |
| Ovarian cyst with twisted pedicle | 21 " | 1½ " " |
| Psychoneurosis and the fear of appendicitis | 17 " | 1⅓ " " |
| Colica mucosa | 5 " | ⅓ " " |
| Ureteral stone | 4 " | ⅓ " " |
| | 1747 cases | |

To the above might well be added a few exceptional cases of inguinal hernia with pain radiating to the iliac region and the more frequent cases of obstruction in the ileocecal region due to neoplasm, tuberculous disease, morbid or post-operative adhesions, abnormal position of the cecum (ptosis, congenital dilatation, etc.); in these cases, however, the pain is generally slight and rarely localized in the iliac fossa.

Examination of this region of the abdomen is of such outstanding importance as to warrant a description here of the systematic procedure to be followed.

The patient being relaxed and recumbent, and the abdomen exposed:

The abdomen should first be examined as a whole, attention being paid to any visible prominence of one abdominal region,

## PAIN IN THE LEFT ILIAC FOSSA.

| Causes | Location of Pain | Kind of Pain | History | Fever and Leucocytosis | Menstruation. | Vaginal and Rectal Palpation. |
|---|---|---|---|---|---|---|
| Salpingitis. Pelvic suppuration. | Iliac and pelvic | Dull pain, brought on especially by vaginal palpation | Gonococcic infection | + | Painful or wanting Leucorrhea | Tender adnexal mass. |
| Dysmenorrhea. | Especially pelvic, with iliac radiation | Of colicky type; at onset of *menstrual* periods | Neurotic virgin. | 0 | Difficult and *pain*ful. | 0 |
| Ovarian cyst with twisted pedicle. | Variable, according to size and location of tumor. | Severe, "peritoneal," and intermittent | Former cystic tumor. | Occasionally | Nothing special | Rounded, elastic, sometimes fluctuating tumor. |
| Extra-uterine pregnancy. | Especially pelvic. | Absent or slight up to the time of rupture. | *Amenorrhea* Signs of pregnancy | Exceptionally. | Amenorrhea. | Tender mass involving adnexa on one side. |
| Sigmoiditis. | Definitely iliac. | Severe, with skin hyperesthesia, "boardlike" abdominal rigidity, *etc*, "*left-sided* appendicitis" | Enterocolitis, dysentery, malaria, or mercurial poisoning. | + | Nothing special. | 0 |
| Cancer of sigmoid. | Definitely iliac | Sometimes absent aside from obstructive attacks | Progressive constipation with attacks of obstruction | 0 | Nothing special | Sometimes involvement of pelvis |
| Stone in ureter. | Variable, from kidney to pelvis, sometimes colic of renal type | Sometimes slight or absent, being elicited only by deep palpation. | *Nephrolithiasis* or pyelonephritis | 0 | Nothing special. | 0 |
| Inguinal hernia. | Sometimes iliac, but more typically inguinal, radiating over abdomen | More marked in standing posture and after exertion | Strain. Weakness of abdominal wall | 0 | Nothing special | Inguinal palpation Inguinal hernia |

be unilateral—hemianesthesia or hemidysesthesia—an examination should be made for the customary stigmata of the neuroses (hysteria). If the disturbances show a metameric distribution corresponding to one of the zones of Head, the condition may be regarded as a cutaneous expression of some deep-seated visceral inflammatory process

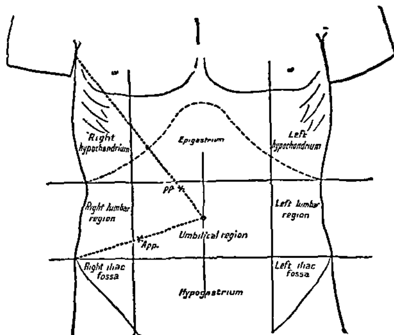



Fig 793—Topography of the abdomen *pp* The pancreatic point *App.* The appendiceal point

Palpation is the chief factor in the examination of this region, and too much care and attention cannot be given to its execution. Both superficial and deep palpation should be practised. In the acute, febrile, peritoneal stages the palpation should be particularly light and cautious It should be carried out with the tips of the fingers, in a gradual manner. Sudden, forcible palpation always excites a defensive reaction of the abdominal wall, even in the normal subject and in the absence of all pain, the abdominal muscles contract, form a barrier, and resist depression by the palpating hand—a condition particularly to be avoided.

due to meteorism, pus accumulation, or a tumor, and noting whether the two sides of the abdomen show different motion in respiration, as might occur through inhibition of one side of the diaphragm because of an underlying process of peritonitis. One

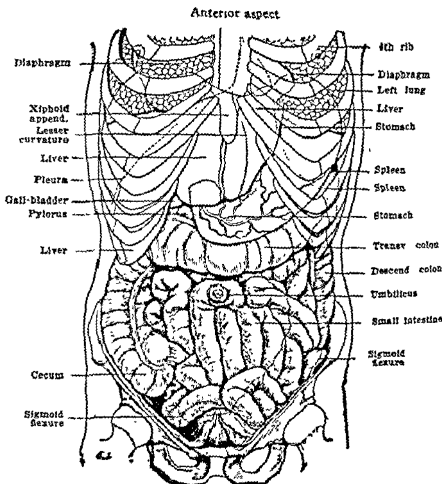



Fig 792—General topographic features of the abdomen (*T Jonnesco, in Poirier and Charpy*)

should also note whether respiration or coughing awaken any localized pain, and if so, at what point

**Skin sensation** should be tested on the two sides by drawing the finger over the skin, pinching, and the application of heat and cold Any existing anesthesia, dysesthesia, or hyperesthesia should be noted. Where such sensory disturbances are found to

regions manifestly free of pain at the time If the subject is well relaxed, the several abdominal regions can be depressed more or less deeply without meeting with too much resistance, often with the result that, by slow, "coaxing," gradual pressure the deep-lying organs may be palpated In the presence of some inflammatory visceral disturbance, and particularly in appendicitis, the palpating finger encounters over the site of pain a firm resistance or insuperable reflex muscular contraction, constituting the so-called "board-like rigidity" This sign, which

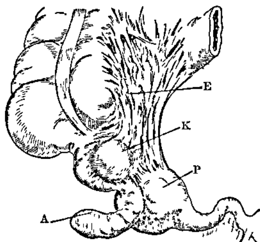



Fig 795—Combined appendicitis and adnexitis The appendix (*A*) is adherent to the ampulla (*P*) of the Fallopian tube which is closed and distended with pus The inflammatory cyst (*K*) is partly covered over by the omentum (*E*) (*Bérard*)

is never absent, is the earliest and perhaps the most reliable indication of appendicitis.

Its exact features must, however, be carefully noted, in some nervous, pusillanimous subjects there may occur a general abdominal rigidity preventing palpation of any of the regions of the abdomen, and under these circumstances the rigidity is devoid of all diagnostic value The same is true of the muscular contraction induced by unduly rough palpation *But whenever a correctly conducted palpation, after noting the absence of undue resistance in other regions of the abdomen, meets with rigid contracture in some*

The patient being well extended in recumbency and eased in mind, with his legs, if possible, slightly flexed, palpation is begun and the various regions of the abdomen (right and left iliac

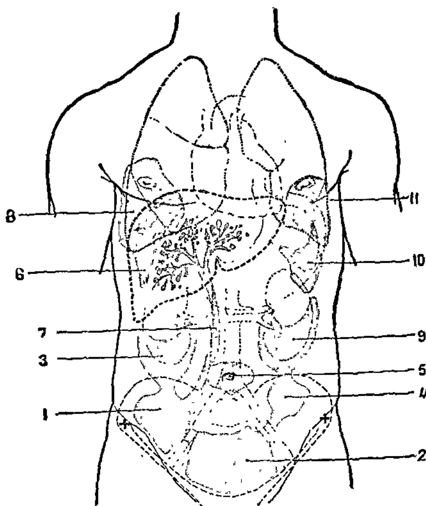



Fig 794—Topographic features of abscesses of appendiceal origin in the order of their frequency *1* The commonest type of abscess, in the right iliac fossa *2* Pelvic abscess. *3* Retrocecal and prerenal abscesses *4* Abscess in the left iliac fossa *5* Mesoceliac and infraumbilical abscesses *6* Right subphrenic abscess *7* Portal and intrahepatic suppuration. *8* Right-sided suppurative pleurisy *9* Left-sided prerenal abscess *10* Infrasplenic abscess *11* Left-sided suppurative pleurisy (*Kelly*).

fossæ and hypochondriac regions, hypogastrium, and umbilical region) lightly and gradually depressed, beginning with the

The foregoing examination of the right iliac fossa should be supplemented with the three following procedures:

(*a*) Manual and bimanual palpation of the right lumbar region for the detection of any existing pus tracks in retrocecal disease, to exclude the possibility of renal disorder, etc.

(*b*) Vaginal palpation in women for the detection of diseased

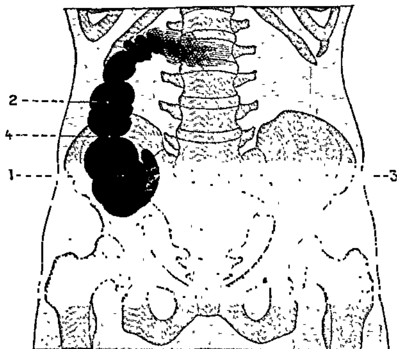



Fig 796—Radiographic picture of the cecum and appendix twelve hours after ingestion of bismuth magma The cecum (*1*) and the ascending colon (*2*) appear dilated and segmented by the constricting bands due to pericolitis The appendix (*3*) is seen occupying a latero-internal position (*Bérard*)

adnexa and pelvic infiltration of appendiceal origin, and rectal palpation in males for prostatitis, ureteral involvement, and extensions of appendiceal suppurative foci into the pelvis.

(*c*) *Gentle* examination of the psoas muscles by flexion, extension, abduction, and adduction of the lower limbs against a slight degree of resistance. Where the psoas is involved, *e.g.*, if there is

*definite area, the result is to be considered pathognomonic evidence of a subjacent inflammatory visceral disorder.*

By the same proceeding of palpation with one finger one should endeavor to ascertain the point of maximum pain. The diagnostic significance of the so-called *McBurney's point* is well known.

Palpation sometimes leads, moreover, to the detection of a mass of varying size, sensitiveness, and evenness of outline, such as an accumulation of feces, an abscess, or a tumor, thorough examination of which will reveal its actual nature.

"On the second, third, or fourth day of acute appendicitis, palpation over the right iliac fossa reveals a doughy condition or broad area of induration which spreads out laterally, is apparently connected with the abdominal wall, and forms a species of thick, *hard* shield This condition, if it possesses and retains the characteristics just referred to, is an outward expression of the 'walling off' process which has set in and gradually extended about the diseased area; such being the case, its presence is actually of favorable import

"About the fifth or sixth day the temperature recedes and drops more or less rapidly to normal, and the pulse rate decreases *in parallel fashion; the hard 'shield,' which has by this* time frequently extended over a broad surface, now ceases to enlarge and becomes still harder in its central portion, while softening and yielding at its periphery, and appears less and less *tender on palpation*" (Lejars, *Chirurgie d'urgence*, p 496)

Finally, it is of advantage, though not absolutely indispensable, to practise "decompressive" palpation, which consists, after more or less deep pressure at some region of the abdomen, in suddenly removing the compressing finger, so that abrupt release of pressure results Sometimes it is noted that, whereas pressure has been relatively painless, decompression causes much pain This seems, to all appearances, to be a reliable indication of inflammation of the peritoneum beneath the palpated area

**Percussion** may prove of great service in the detection of an abnormal, flat area in certain cases in which satisfactory palpation is practically impossible. It may, furthermore, be availed of as a gentle method of palpation

Thus, the reader looking up in a certain chapter a variety of pain commonly described as being localized in a certain region may wonder at its absence from that point and at finding it elsewhere Again, some causes of pain may be found referred to in

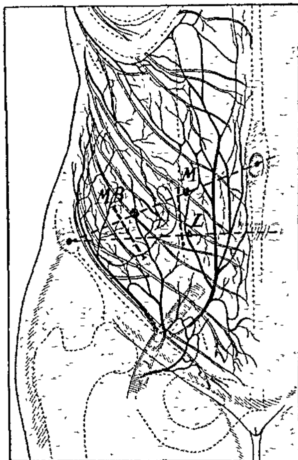



Fig 797—Vessels and nerves of the anterior abdominal wall projected upon the ceco-appendicular region *MB* McBurney's point. *M* Morris's point *L* Lanz's point The dotted line pointing to *MB* shows the line of the McBurney incision The dotted line *ML* shows the Jalaguier incision (*Bérard*)

two different chapters (ovarian cyst with twisted pedicle, ectopic pregnancy, etc) because they are equally common on the right and the left sides of the body.

appendiceal inflammation adjoining this muscle, such an examination of the latter is a painful procedure.

In conclusion it may be added that it is always well to examine the right iliac fossa under anesthesia just before the operation

"Before operating, one should never neglect to conduct a *final examination upon the well anesthetized patient* in a state of complete muscular relaxation. Frequently the yielding of the abdominal wall, which is no longer rigid, will lead to the detection of a more or less distinct prominence in the right iliac fossa, or upon *oblique inspection,* a marked lack of symmetry of the two lateral halves of the abdomen may become apparent.

"Examination by palpation will afford more exact information; generally the examiner will find one of the following conditions Either a *definitely fluctuating pocket,* tense, circumscribed, and sharply defined at its mesial border; a *thick sausage-like mass,* compact, indistinctly or partially fluctuating, or with nodular surface and poorly defined borders below and toward the median line, or a *small, hard mass,* rounded or nodular, non-adherent and readily mistaken for the appendix itself.

"Sometimes the tumor noticed in the waking state will seem to have almost completely disappeared It may be added that if, when the patient has been anesthetized, iliac palpation continues to give the impression of a *diffuse doughy condition,* while the abdomen fails to recede and remains prominent and tense, the previous apprehensions of generalized peritonitis are to a singular degree confirmed." (Lejars)

The following summary reflections, of general application in the diagnosis of abdominal disorders, are borrowed from Cabot (*"Differential Diagnosis"*)

Though it seems judicious and is in accord with current practice to differentiate the exciting causes of the various localized and diffuse abdominal pains, as a matter of fact such distinctions do not always hold good. Disorders such as appendicitis, theoretically attended with pain in the right iliac fossa, may very readily cause pain localized even above the waist line. Again, lead poisoning, which ordinarily gives rise to distinctly diffuse pains in the last-named region, may instead readily cause a much more circumscribed pain.

| | | | | | | |
|---|---|---|---|---|---|---|
| **Dysmenorrhea.** | Pelvic. | Of colicky type, at the onset of menstruation | Neurotic virgin | 0 | Painful | 0 |
| **Extra-uterine pregnancy.** | Chiefly pelvic | Absent or slight up to the time of rupture | *Amenorrhea* Signs of pregnancy | *Sometimes* | *Amenorrhea* | *Tender adnexal tumor* |
| **Stone in ureter.**[1] | Variable, from kidney to pelvis, sometimes colic of renal type | Sometimes slight or absent, being elicited only by deep palpation | Nephrolithiasis or pyelo-nephritis. | Generally absent | Nothing special | 0 |
| **Psychoneurosis.** | Generally that of appendicitis | No true rigidity, no pain on "decompression" | Neurotic Previous cases of appendicitis among patient's associates | 0 | Nothing special. | 0 |
| **Inguinal hernia.** | Sometimes in iliac fossa, but rather along inguinal canal, radiating over abdomen | More marked in standing posture and after exertion than in recumbency and at rest. | Strain Weakness of abdominal wall | 0 | 0 | Inguinal palpation, hernia, or omentum, or broadened ring. |

[1] Radiography may sometimes disclose the presence of a stone in the ureter

## PAIN IN THE RIGHT ILIAC FOSSA.

| Causes | Location of Pain | Kind of Pain | History | Fever and Leucocytosis | Menstruation | Vaginal and Rectal Palpation. |
|---|---|---|---|---|---|---|
| Appendicitis. | Right iliac fossa<br>Maximum pain at McBurney's point. | Often diffuse, then localized<br>Hyperesthesia of abdominal wall<br>Muscular contraction (rigidity) | Sometimes previous attacks, indigestion, constipation, vomiting. | + | Nothing special. | Sometimes tender mass above and to the right (above the pelvis) |
| Typhlocolitis. Pericecal tuberculosis. | Right iliac fossa, colon, and sometimes left iliac fossa | Colicky pain often generalized over the entire abdomen or extending along the colon. | Neurosis<br>Constipation<br>Muco-membranous bowel disturbance. | 0<br>Terminal | Nothing special. | 0 |
| Salpingitis. | Pelvic, lower than in preceding condition | Little or no rigidity.<br>Tenderness more especially on vaginal palpation. | Gonorrheal infection. | + | Painful or lacking<br>Leucorrhea. | Tender adnexal mass. |
| Ovarian cyst with twisted pedicle. | Variable according to size and position of cyst | Severe; sometimes intermittent. | Long-standing cystic tumor. | Sometimes | Nothing special | Cystic tumor, rounded, elastic, and sometimes fluctuating |

# INSOMNIA.

[In, *negative;* somnum, *sleep;* *deprivation of sleep.*]

---

Insomnia or *agrypnia* consists in a more or less complete and lasting inability to sleep. It occurs in all grades, from simple *hyposomnia,* characterized by shorter, lighter, more restless, and less refreshing sleep than usual, to the obstinate and inveterate *complete insomnia,* sometimes attended with a very unfavorable prognosis.

The causes of sleeplessness are many For practical purposes they may be divided into the three following groups:

*Insomnia due to pain*

*Insomnia due to excessive nervous excitability.*

*Insomnia due to circulatory or respiratory disturbances*

**Insomnia due to pain** may be the result of *any kind of pain,* of whatever situation and nature, including the most varied forms of *neuralgia, arthralgia,* and *visceralgia.* To attempt to enumerate all these causes would be tiresome to the reader and plainly superfluous At the most it will be well to call attention to the fact that some "pain insomnias," when carefully traced, lead to the discovery of certain "algias" which recur at night and are of special clinical significance, such as the osteocopic pains of syphilis, sometimes the neuralgias of tabes, and more frequently the myalgias and arthralgias of gouty subjects

Again, it should be noted that *itching* or *pruritus* (see *Itching*) of whatever cause (parasitic or toxic) may be the source of highly obstinate insomnia. When correctly traced, this symptom leads to the detection of numerous "incomplete" cases of diabetes, uremia, and cholemia.

Lastly, it may be pointed out that the insomnias due to special sense hyperesthesia and the psychosplanchnic neurosis merge insensibly with the next succeeding group, affording a good illustration of the fact that clinical conditions, with their infinitely numerous variations, always tend to pass beyond the

*In a general way, when the practitioner is seeking to find out the* probable cause of a pain in the abdomen, *he should be guided by the following rules*:

1. *First of all he should suspect the gastrointestinal tract,* and if the most commonplace disorders, such as constipation and colitis, can be excluded, he should think especially of appendicitis, peptic ulcer, *neoplasm of the stomach or large bowel,* and the ultimate consequences of these conditions, such as peritonitis and intestinal obstruction.

2. Next he should suspect (in women) the generative tract—*salpingitis, ovarian cyst, uterine fibroids,* and *ectopic pregnancy.*

3. The gall-bladder and bile-ducts should be particularly investigated in persons over middle age

4. The urinary tract, especially in old men and young girls, comes next in the list of causes of abdominal pain.

Clinical examination, the history, palpation, blood examination, uranalysis, fluoroscopy, and cystoscopy are the most serviceable aids in reaching a diagnosis.

* * *

The frequent concurrence of appendicitis and cholelithiasis or painful congestion of the liver—manifested by two typical sites of tenderness, *viz*, right iliac fossa and right hypochondrium—sets up a diagnostic problem sometimes quite difficult to solve. Are appendicitis and gallstones both present? Is the tenderness at McBurney's point not merely a reflex radiation from cholelithiasis, of sympathetic origin? or conversely?

Some differential signs—upon which one should not place too much reliance—have been described by various authors:

1. In cholelithiasis, the fingers pushed in beneath the costal margin at the gall-bladder point prevent inspiration (Parturier and Murphy). 2. Finger pressure on the right between the two heads of the sternocleidomastoid elicits the sensitiveness of cholelithiasis, while in appendicitis it is without effect (Chauffard).

Practical conclusion: When in doubt, do not abstain—it is better to have the patient undergo an appendectomy than expose him to serious complications.

acute rheumatism and pneumonia. Others seem to be *true toxic-infectious insomnias,* of dyscrasic origin, due to the action of the toxins of infection on the nerve centers, as in the early insomnia of typhoid fever, of grippe, and of erysipelas. Lastly, an additional group, especially related to convalescence, appears to be dependent upon neurovascular weakness, *e.g.,* the *insomnias of anemic and asthenic cases* (starvation, convalescence, sequelæ to infections, etc.).

**Insomnia due to circulatory or respiratory disturbances.**—*Insomnia dependent upon some cardiopulmonary affection.*—This is the insomnia of heart failure, of lost compensation, of cardiopulmonary disorders causing cough and dyspnea, of asthma, of chronic bronchitis, etc. The causes are many and outstanding, *viz.*, cough, dyspnea, and toxic influences.

Thus, *insomnia* is a very common symptom and consequently one of very restricted diagnostic value, except possibly in the psychoneuroses This does not apply, however to its causal diagnosis, for it is from such a study of its cause that the basis for rational and effectual treatment can be found. A patient with heart weakness is made to sleep by restoring circulatory balance; a coffee fiend by withdrawing the drug, and a syphilitic subject by specific treatment.

Even with reference to much less definite groups of cases, however, rational use of special hypnotic remedies depends upon a partial knowledge of the pathologic physiology of insomnia, and as an illustration of this fact it seems of interest to consider summarily the respective indications of chloral hydrate and of morphine in this class of cases

## INDICATIONS AND CONTRAINDICATIONS TO CHLORAL HYDRATE AND MORPHINE FOR HYPNOTIC PURPOSES.

Chloral hydrate and morphine are probably—and with reason—the two most commonly employed hypnotics. They should not, however, be thought of as being interchangeable While they may sometimes be administered with advantage in combination, they actually meet wholly different indications and should be prescribed only for definite reasons.

*Chloral hydrate* and *morphine* appear to be *directly acting hypnotics,* i.e, drugs inducing sleep by a selective, direct action upon

group limitations within which the physician, for his convenience, endeavors to confine them.

**Insomnia due to abnormal excitability of the nervous system.**—Such nervous overexcitability may be the result:

1 *Of some organic change in the nervous system* (type condition: **meningitis**).

2. *Of an overexcitation of functional origin* (type condition: **psychoneuroses**).

3. *Of an intoxication or infection* (type conditions: **caffeinism** and **typhoid fever**)

In the *first group* are included the insomnias of meningitis, brain tumor, general paralysis, and cerebral syphilis. In all these cases the symptomatic combination of headache and insomnia exists

The *second group*, that of the *psychoneuroses*, is much more frequent. In it are comprised the so-called "nervous" insomnias dependent upon overwork, excessive ideation, worry, mental excitement (irritable weakness, emotivity, emotional impressions), mania, psychoneuroses, hysteria, neurasthenia, obsession, phobia, and anxious states "In acute attacks of psychosis," states Régis, "insomnia is one of the first symptoms to appear; it is manifested particularly in restlessness, dreaming, and nightmares. On the other hand, restoration of the ability to sleep towards the close of a period of mania or melancholia is of excellent prognostic import" In chronic mental diseases insomnia is uncommon, except among insane subjects harboring hallucinations or cenesthetic illusions. Insomnia as a symptom should receive careful treatment in all psychoneurotic states: A patient able to sleep is already half-cured under these circumstances.

The *third group*, the insomnia of *toxic* and *infectious states*, is more complex.

In this group some forms of sleeplessness seem to be instances of *true toxic insomnia* due to direct stimulation of the cerebral cells; among these are the insomnias due to abuse of tea, coffee, alcohol, tobacco, morphine, cocaine, etc.

As for the *infectious* and *post-infectious insomnias*, their mode of production is unquestionably much less simple. Some appear to be *algic insomnias* dependent upon some predominant painful disturbance (headache, joint pain, or pain in the side), as in meningitis,

acute rheumatism and pneumonia. Others seem to be *true toxic-infectious insomnias*, of dyscrasic origin, due to the action of the toxins of infection on the nerve centers, as in the early insomnia of typhoid fever, of grippe, and of erysipelas. Lastly, an additional group, especially related to convalescence, appears to be dependent upon neurovascular weakness, *e.g*, the *insomnias of anemic and asthenic cases* (starvation, convalescence, sequelæ to infections, etc.).

**Insomnia due to circulatory or respiratory disturbances.**—*Insomnia dependent upon some cardiopulmonary affection*.—This is the insomnia of heart failure, of lost compensation, of cardiopulmonary disorders causing cough and dyspnea, of asthma, of chronic bronchitis, etc. The causes are many and outstanding, *viz.*, cough, dyspnea, and toxic influences.

Thus, *insomnia* is a very common symptom and consequently one of very restricted diagnostic value, except possibly in the psychoneuroses. This does not apply, however to its causal diagnosis, for it is from such a study of its cause that the basis for rational and effectual treatment can be found. A patient with heart weakness is made to sleep by restoring circulatory balance, a coffee fiend by withdrawing the drug, and a syphilitic subject by specific treatment

Even with reference to much less definite groups of cases, however, rational use of special hypnotic remedies depends upon a partial knowledge of the pathologic physiology of insomnia, and as an illustration of this fact it seems of interest to consider summarily the respective indications of chloral hydrate and of morphine in this class of cases.

### INDICATIONS AND CONTRAINDICATIONS TO CHLORAL HYDRATE AND MORPHINE FOR HYPNOTIC PURPOSES.

Chloral hydrate and morphine are probably—and with reason—the two most commonly employed hypnotics They should not, however, be thought of as being interchangeable. While they may sometimes be administered with advantage in combination, they actually meet wholly different indications and should be prescribed only for definite reasons.

*Chloral hydrate* and *morphine* appear to be *directly acting hypnotics*, i.e., drugs inducing sleep by a selective, direct action upon

the nerve cell. This constitutes, however, about the only property they have in common. Indeed,

1. *Opium and its derivative, morphine, exert,* in moderate dosage, —as Sydenham had plainly noted—a *tonic action on the heart;* under their influence the heart beats develop increased amplitude and power, the blood-pressure rises, and circulation through the viscera becomes more active.

*Chloral, on the other hand, is a cardiovascular depressant;* under its influence the heart beats become weaker and less frequent, the blood-pressure is reduced, and visceral circulation is less active

2. *In the first stage,* at least, *opium and morphine produce evidences of cerebral stimulation* (a property availed of by morphinists), in all likelihood through hyperemia of the brain and meninges and direct nervous action. The sleep induced is frequently associated with dreaming; sometimes it presents features suggesting the so-called "coma vigil."

*Chloral sleep, on the other hand, is not preceded by any stage of stimulation;* it is in all respects comparable to normal sleep as to general features and duration

3 *Finally, morphine is an analgesic agent of the first order,* being the type of the pain-relieving drugs

*Chloral, on the other hand, is neither analgesic nor anesthetic,* pain prevents chloral sleep from coming on, while loud noises awaken the sleeping patient.

Such are the more salient differences between chloral hydrate and morphine *Their respective indications and contraindications* are logically based on these differences in action

*Morphine, a cardiac and vascular stimulant, at least temporarily a stimulant of the brain functions, and an analgesic agent of the first order, is especially indicated in insomnia dependent upon or associated with neurovascular weakness or some painful disorder*

Such being the case, it is serviceable in *pain insomnia,* generally due to neuralgia or visceral pain, as well as tabes dorsalis, cancer, etc. In these cases, however, in order to obviate or postpone as much as possible the risk of morphine habit, it is well not to resort to it until after the entire list of pure analgesics, such as acetphenetidin, antipyrin, exalgin, salipyrin, etc., has been exhausted

In the *insomnia of anemic or asthenic subjects* (inanition, convalescence, typhoid fever, pneumonia, etc.) or of persons *with weak heart action* or *low blood-pressure*, morphine, with or without the addition of heart tonics, remains the hypnotic remedy of choice.

In these cases, on the other hand, chloral hydrate proves ineffectual or even usually does harm.

In the so-called *nervous insomnia*, however, *dependent upon overwork, excessive ideation*, worry, mental excitement, mania, alcoholism, meningeal congestion, and high blood-pressure, morphine not only proves ineffectual, but is frequently even dangerous. *Chloral hydrate is the hypnotic of choice* in these cases.

Lastly, there occur a large number of hybrid clinical species, and various "mixed" insomnias, which warrant combined use of the two drugs to some extent.

Such, for example, is the insomnia of overworked anemic subjects, the painful insomnia present in high blood-pressure (neuralgia in a case of arteriosclerosis), etc. Under these circumstances the combination of chloral with morphine, while it doubtless fails to afford an ideal pharmacodynamic procedure, constitutes a logical solution of the problem of sleep induction

Were it necessary to summarize in one concise sentence the above considerations, it might be stated that:

*Opium and morphine are indicated in insomnia associated with neurovascular weakness or pain, chloral hydrate, in insomnia related to neurovascular overactivity, without pain.*

# ITCHING.

The least inaccurate definition of itching or pruritus would appear to be that of Jaccoud, *viz.*, pruritus is the sum of the subjective sensations which awaken the desire and need of scratching

The frequent association of pruritus with vasomotor disturbances, as in dermographia, has led to the view that its cause may be an organic or functional disturbance of the sympathetic nerves.

Many different classifications of pruritus, based on its pathogenesis and clinical features, have been formulated. The best, for practical purposes, appears to be the following·

Toxic (and metabolic) pruritus.
Dermatosic pruritus (due to skin lesions).
Parasitic pruritus.
Neurotic pruritus.

**Toxic pruritus** or itching includes all those forms of pruritus in which the cause seems to be actually some change in the tissue fluids or blood, whether this change be metabolic (autotoxic) in nature, or a true intoxication of food or drug origin (exotoxic).

**Autotoxic metabolic pruritus** is extremely common. It is met with in *diabetes* (*diabetic pruritus*), in *gout*, in *uremia*, in *stercoremia* (constipation), in *cholemia* (itching in jaundice), in *arteriosclerosis* (*senile pruritus*), in dyspepsia, and in dysmenorrhea Its outstanding feature is plainly the conception of an altered humoral state consequent upon the insufficiency of the liver and kidneys which is characteristic of most of the above mentioned disorders.

**Pruritus of alimentary origin** is no less common, and the large number of the persons predisposed to it is well known The more particularly prurigenous articles of food are crustaceans and other shell fish, preserved and salted meats, game, stale fish, spices, an excessive meat diet, fermented cheeses, and strawberries A partial insufficiency of the liver and kidneys seems to be at the bottom of these various types of food intolerance. Possibly

the factor of anaphylaxis is also concerned, as in the succeeding group of cases.

**Pruritus of pharmaceutic origin**, due to *coffee, tea, alcohol, belladonna*, cocaine, antipyrine, mercury, bromides, chloral hydrate, opium and its derivatives, and the balsamic remedies. Abuse of these drugs in some persons, and their use in ordinary dosage in many, may be the source of itching with or without actual skin disease.

**Pruritus of hydatid origin** should also receive recognition in this group.

**Dermatosic pruritus** comprises all the skin affections which give rise to itching. The commonest are: Prurigo, urticaria, lichen, eczema, mycosis fungoides, chicken-pox, seborrhea, hyperidrosis, Duhring's dermatitis herpetiformis, the ringworms, etc. One cannot resist the temptation to reproduce *in extenso* Brocq's lecture on the topic of pruriginous skin disorders:

"I am reviewing for you briefly how one may understand and classify the pruriginous dermatoses which fall into the group I term that of the simple skin reactions with pre-eruptive pruritus (Jacquet)

"1 When a patient is seized with pruritus and scratches himself, the integument, even though exposed to the trauma of scratching and rubbing, may retain its normal appearance, showing no structural change appreciable to the naked eye, *i.e.*, no eruption. One may thus say that *it is not reacting in a visible manner* This constitutes *simple pruritus* or *pruritus sine materia.* This is the so-called *idiopathic pruritus,* a rather frequent condition in private practice, especially among neurotics; *senile pruritus* belongs in this group

"2 Under the influence of scratching and rubbing, the skin may more or less rapidly exhibit a changed appearance; it may assume a slightly brownish tint; the creases in it become enhanced and more readily visible, deeper, and cross one another in diamond-shaped figures of varying regularity; the appearance at first becomes velvety, then rugose, owing to accentuation of the dermal papillæ and of the epidermis, histologically, indeed, there is produced a very marked hyperacanthosis. The process

may stop at this point, as is nearly always the case when the pruritus is of general distribution; when it is circumscribed, however, the skin lesions undergo further development, suggesting at first species of papules arising through papillary and epidermal hypertrophy, and later infiltrated, thickened, cross-hatched plaques, more or less scaly and excoriated. These are the changes characteristic of *simple lichenification,* a process which, like pruritus itself, may be either diffuse or circumscribed; and as I showed twenty-two years ago while attempting a complete differentiation of these morbid types, these are lesions of a purely traumatic origin, which may be either *primary, i.e.,* show initial development on a healthy skin, or *secondary, i.e.,* become superimposed upon any other pre-existing pruriginous skin disorder. When primary, the condition constitutes the *lichen simplex of the older authors,* or, in our own nomenclature, *diffuse pruritus* or *circumscribed pruritus with lichenification.*

"3 Under the influence of scratching and rubbing the skin may react by the production of an ordinary *urticaria;* it may react with the so-called *urticaria papulosa,* characterized by small, papular lesions, and the resulting sequence of changes tends toward the appearance of prurigo (see below); again, it may react with *urticaria bullosa,* and the resulting sequence of changes tends toward the appearance of dermatitis multiformis (see below).

"4. Under the influence of scratching and rubbing, especially when the pruritus is localized on the inner aspects of the fingers, the patient may note almost immediately the formation of certain elevations of the epidermis filled with citrine, clear, serous fluid and free of surrounding redness, the result being that the skin appears as though peppered with boiled sago grains, closely aggregated and sometimes so confluent as to form rather extensive areas of raised epidermis, almost always discrete, or merely in apposition. This is the clinical picture for which the term *dysidrosis* should properly be reserved; frequently, however, it is present in combination with the following type of disturbance, whence unfortunate mistakes are apt to result.

"5. Under the influence of scratching and rubbing, there develop on the skin, sometimes without redness, but nearly always with a more or less striking erythema, fine vesicles of un-

equal size, of the average size of a pinhead, and which dot the epidermis in highly irregular fashion. The best plan for observing them plainly is to first dry the skin either with a piece of fine cloth or with cotton impregnated with sulphuric ether, then apply over the affected surface a piece of cigarette paper, over which is placed a piece of glass to exert pressure Serous fluid from the vesicles is then seen through the glass to ooze out and be absorbed by the paper, thus showing very clearly the shape and arrangement of the little vesicles. If the latter have not yet ruptured, one need merely make a few very light strokes with a curette and then apply the cigarette paper and pressure glass To this objective morbid condition, definitely characterized by the peculiar fundamental skin lesion just referred to, I apply the term *eczema vulgaris, true vesicular eczema*, or *amorphous eczema*

"6 Under the influence of scratching and rubbing there develop minute lesions of a rather bright red color, slightly elevated above the surrounding skin surface, exhibiting at their center a slight lifting up of the epidermal layer by citrine serous fluid, *i e.*, a small vesicle These lesions may be scattered here and there in complete disorder, especially on the extremities, but exhibit a marked tendency to become agminated and confluent, thus giving rise to red patches, dotted with minute vesicles similar to those of the preceding type and oozing more or less freely. This is the disorder to which the term *papulovesicular eczema* is peculiarly applicable It is made up of a number of transitional stages which insensibly merge the true, common or amorphous vesicular eczema with the true prurigoes to be next described

"7. Under the influence of scratching and rubbing, the skin reacts with bright red, acuminate, more or less urticarial papules, exhibiting at their apices a slight tendency to elevation of the epidermal layer by a little citrine serous fluid. As the attendant itching is very marked, these urticarial papulovesicles (Tommasoli's seropapules) are nearly always found ruptured by the patient's finger nails, where, however, the lesion is permitted to run its course without traumatic interference, there arises spontaneously at its summit a minute brownish-yellow crust formed through desiccation of the little apical vesicle. Such is the characteristic fundamental lesion of *prurigo*. As I already stated in

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discussing the differential diagnosis of one of our cases, if these eruptive units remain separate and discrete, the morbid type known as *prurigo simplex* exists; if they show a tendency to come together in clusters and form eczematized and lichenified plaques, the condition is that known as *Hebra's prurigo;* if they are very large, the condition present is *prurigo ferox Vidali.*

"8. Following scratching and rubbing there may be produced in certain patients only a more or less marked lichenification of various extent, together with acute out-croppings of eczema vesicles. E Besnier long ago classified this disorder among the 'diathetic prurigoes.' According to the nomenclature personally adopted, it cannot be spoken of as a form of prurigo, since it fails to exhibit the fundamental urticarial papulovesical characteristic of this group For it I shall therefore retain the term *pruritus with lichenification and eczematous transformation.*

"9. Under the influence of scratching and rubbing, the skin may finally react in a much more complex manner. In some places there form patches of erythema, elsewhere urticarial lesions, elsewhere, either on healthy skin or over pre-existing patches of erythema, vesicles or blebs of varying size, and sometimes even pustules. These various eruptive types may be simultaneously present in the same individual, constituting the multiform eruption *par excellence;* they may instead occur in succession, one eruptive outburst being, *e.g.*, urticarial, another erythematous, another erythematovesicular, another bullous, another actually multiform, etc. Furthermore, the various eruptive lesions may be scattered in disorderly fashion; they may be grouped together, and suggest either herpes vulgaris or the circinate lesions of ringworm, in which event the term *dermatitis herpetiformis* (Duhring) is particularly applicable The clinical group as a whole should be termed that of *dermatitis multiformis*

"Such, briefly summarized, is the vast series of the pruriginous skin disorders with pre-eruptive pruritus (Jacquet) belonging to the group of the primary skin reactions.

"It should be added, however, that under the influence of itching and the attendant scratching, other eruptions, which cannot, for the present at least, be classed simply among the skin disorders with pre-eruptive itching, may likewise develop with extreme ease and

rapidity. In the front rank among these should be mentioned those peculiar disturbances, intermediate between eczema and psoriasis, which have been the subject of such extensive discussion of late and to which we have applied the term *psoriasiform parakeratoses;* now, among these psoriasiform parakeratoses there is one particular form which is frequently seen to develop under the circum-

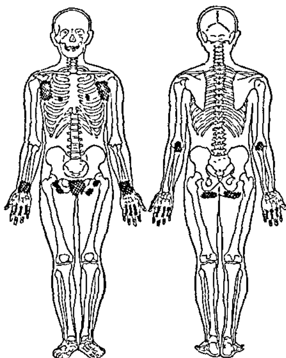


Fig 798—Scabies Places of election for burrows None are ever noted on the face or scalp

stances alluded to. This variety is chiefly characterized, objectively, by the presence of patches of varying extent of a more or less bright red color, sometimes pale, sometimes rather dark, scaly, and over which are formed vesicles similar to those of true vesicular eczema This condition is therefore actually deserving of the appellation *eczema;* it is what most authors term *seborrheic psoriasiform eczema,* but what I have referred to as *eczematized psoriasiform parakeratosis,* wishing thereby to imply that in many instances this clinical condition is in no wise related to seborrhea."

Along with these various forms of dermatosic pruritus should be mentioned the itching due to varicose conditions of the lower extremities. These are always *accompanied by trophic disturbances.*

The same applies to the localized forms of pruritus, such as vulvar or perianal pruritus due to some local uterine, vaginal (leucorrheal), urethral, anal or perianal (fistula or hemorrhoids) discharge.

**Parasitic pruritus** is clinically represented chiefly by *scabies* and the several varieties of *pediculosis.* These conditions should always be kept in mind, though little should be said about them,

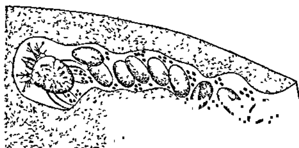


Fig 799—Burrow containing a female itch-mite and her ova

even after the diagnosis is certain. The diagnosis of **scabies**—an important one to render—should be based chiefly on the *transmission of an itching disorder* (the patient "having slept with some one who was frequently scratching himself"), on the *special localization of the itching at the points of election shown in the annexed illustration,* and if **necessary,** by *microscopic identification of the parasites themselves* For the latter purpose one of the little burrows in the skin should be opened up with a needle and the minute white object at the bottom of it removed, *likewise with the needle;* the female parasite is thus secured and may be examined with a hand lens or microscope.

The following description of one of these burrows is reproduced from Sabouraud.[1]

"**The Burrow.**—I am for the first time thus alluding to the familiar burrow of scabies. This is because in any fairly extensive

[1] *Presse médicale,* June 21, 1917.

dispensary practice, the dermatologist will have twenty times made a diagnosis of scabies on the basis of the localizations of the disorder before having searched for *a single* burrow. The burrow is looked for in recent or doubtful cases in which no light is thrown on the condition by the history. What, then, is the scabies burrow? A homely comparison will give an idea of it at once, for every one is familiar, from repeated observation, with the burrow of a mole projecting above the surface of the ground in a field. The burrows of the itch-mite are constructed similarly They are most readily

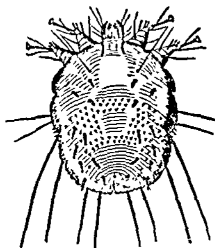


Fig. 800—*Sarcoptes scabiei*, female, dorsal aspect (*R Blanchard*)

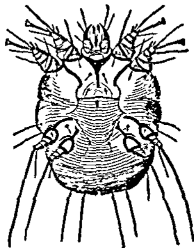


Fig 801—*Sarcoptes scabiei*, female, ventral aspect (*R. Blanchard*).

observed in uncleanly individuals working in dirty liquids, since these liquids, entering the burrows by capillarity, stain them black To see them well when one is not familiar with them, the palm of the hand in the children of the very poor should be selected The burrows may be accurately compared to the outline of the spirochete of syphilis stained with silver nitrate, now a familiar object owing to its frequent reproduction by photography as well as by delineation It appears as a wavy black line. Where the burrow is not blackened with dirt, howerer, it is so hard to see that the observer, in order to remove all doubt, should stain it by placing a drop of ink or tincture of iodine over it, wiping it off a moment later—a simple, but often useful procedure

"The unblackened burrow is even harder to describe than to descry. Let the reader imagine that he has pushed a needle through thick, horny epidermis, *e.g.*, at the finger tip, without

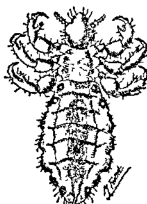


Fig 802—*Pediculus capitis*, male Enlarged 25× (*Brumpt*).

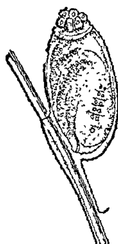


Fig. 803—Ovum of *Pediculus capitis* attached to a hair. Enlarged (*Brumpt*).

drawing any blood. When the needle is withdrawn, the track made by it will be visible, the raised epidermis having been ren-

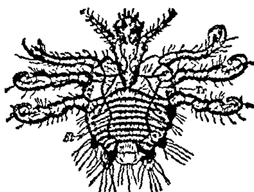



Fig. 804—*Phthirius pubis*. Enlarged 25×. *St.* Stigma *Tr.* Air-duct.

dered dull and whitish; the channel thus made is, however, a straight one, while that of the itch-mite is always wavy. Otherwise, the white, dull condition of the epidermis induced is exactly the same. Such a channel is quite hard to see, and this is what

makes the diagnosis of the disease so difficult, except under hospital conditions. More commonly the lesions of scabies are elevations, papules, or vesicles often opened by scratching, and the long axis of which exhibits the same direction as the skin fold at that point."

The diagnosis of pediculosis (phthiriasis) is similarly based on the situation of the skin lesions (see illustration) and direct examination of the parasite. It should not be overlooked that these para-

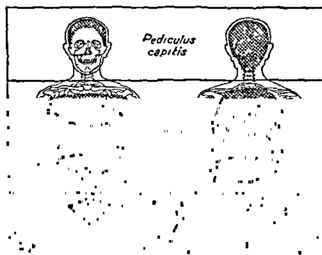



Fig 805—Pediculosis or phthiriasis Areas of election.

sites are not only unpleasant, but also dangerous, being known carriers of many instances of relapsing fever and of typhus fever. The well-known *maculæ ceruleæ* above the pubis, characteristic of pediculosis pubis, should be kept in mind

**Neurotic pruritus** is met with chiefly under the three following circumstances

(*a*) In psychoses, neuroses, exophthalmic goiter, and as a sequel to overwork, sorrow, and severe emotional impressions.

(*b*) In lesions of the peripheral nerves (causalgia).

(*c*) As a reflex manifestation of some deep-seated visceral irritation, as exemplified in the itching of intestinal helminthiasis.

*To recapitulate*: Itching may, for practical purposes, be divided into the following five groups of causes, which account for at least 95 per cent. of all cases:

1. **Parasites:** Scabies and pediculosis; in these cases the diagnosis is based on

(*a*) *Localization of the itching: Head and neck in ordinary* pediculosis; dorsal surfaces of the hands and forearms, anterior aspect of the axillæ, inguinal regions, and penis in scabies; pubic region in pediculosis pubis

(*b*) The skin lesions due to scratching.

(*c*) Direct observation of the parasite concerned with a good hand lens or microscope

2 **Itching skin affections:** Observation of the type of skin disorder present enables the experienced dermatologist to render an immediate diagnosis.

3 **Hepatic and renal insufficiency:** *Cholemia* and *azotemia*

(*a*) Blood-pressure estimation, uranalysis, the presence of other evidences of azotemia (vertigo, cramps, epistaxis, and nycturia), and in particular, *determination of the blood urea* will lead unmistakably to the diagnosis of *azotemia*

(*b*) The *itching attending jaundice* is a familiar symptom One should be able, however, to detect even an early cholemia, of which itching is itself a valuable indication

4. **Metabolic disorders,** in the front rank of which should be placed **diabetes.** Pruritus in certain regions, *e.g*, the *inveterate pruritus vulvæ* of women, is particularly significant. One should never omit examining the urine in a case of pruritus, for four distinct *reasons—sugar, albumin, bile, and acidity.* Glycosuria, nephritis, cholemia, and acidosis are extremely common causes of itching.

5. **Neuropathic states** in which pruritus is an actual cutaneous dysesthesia, accompanied by the usual characteristic evidences of neurosis The diagnosis of neuropathic pruritus should, however, be made only by exclusion, after having systematically eliminated the causes already mentioned, *viz.*, parasites, skin disorders, cholemia, azotemia, glycosuria, and dietetic or pharmaceutic intoxication

## ITCHING.

| Causes | Special Features and Location of Itching | Urine Examination | General Condition Associated Clinical Signs |
|---|---|---|---|
| **Parasitic.** | Scratch marks<br>Special parasite | | |
| **Pediculosis.** | Head and back of neck | 0 | 0 |
| **Scabies (burrows)** | Dorsal aspect of hands and forearms<br>Anterior aspect of axillæ<br>Prepuce, inguinal regions<br>Pubes<br>Nocturnal paroxysms | | |
| **Dermatosic.** | Typical skin affections, such as prurigo, urticaria, lichen, eczema, seborrhea, ringworm, chicken-pox, mycosis, etc | Sometimes albumin if the skin disorder is generalized | 0 |
| **Cholemic.** | Generalized itching *sine materia*, sometimes predominantly on the lower extremities<br>Scratch lesions late in appearing | Sometimes bile pigments<br>Nearly always urobilin, and frequently alimentary glycosuria | Established or incipient jaundice<br>Bradycardia<br>Familial cholemia |
| **Azotemic.** | Generalized itching *sine materia*<br>Sometimes nocturnal paroxysms<br>Scratch lesions late in appearing | Frequently albumin | Evidences of Bright's disease and arteriosclerosis<br>High blood-pressure<br>Headache, vertigo, epistaxis, nycturia, etc |
| **Diabetic.** | Itching often localized at the vulva, or skin folds on flexor surfaces, extremely marked and obstinate | Glycosuria | The usual signs of diabetes:<br>Polyuria, polydipsia, polyphagia, etc |
| **Toxic.** | Generalized itching *sine materia*, or urticarial, or dermatitis medicamentosa (Ex · Exfoliative dermatitis of mercurial origin) | 0<br>Or transitory albuminuria<br>Or transitory urobilinuria | Dietetic or pharmaceutic intoxication, more or less obvious<br>Yields more or less quickly to removal of the cause |

**ITCHING** (*continued*).

| CAUSES. | SPECIAL FEATURES AND LOCATION OF ITCHING | URINE EXAMINATION | GENERAL CONDITION. ASSOCIATED CLINICAL SIGNS |
|---|---|---|---|
| **Neuropathic.** | General or local itching *sine materia* | 0 | (*a*) Psychopathic disorder, neurosis, exophthalmic goiter, emotions, overwork, etc. (*b*) Peripheral neuritis (causalgia) (*c*) Remote visceral irritation (helminthiasis). |

# JAUNDICE (ICTERUS).

The term icterus is applied in all cases in which—whether the urine and stools are or are not affected—the conjunctivæ and skin exhibit a yellow or yellowish hue. In this work the word icterus, in conformity with its etymologic derivation (from ἴχτερος, jaundice), will be taken as synonymous with jaundice and as being free of any implication that the discoloration is of biliary, hepatic, or other origin

*Icterus* or *jaundice* may be caused:

1. By retention and reabsorption of the bile and of the normal biliary pigments: *Hepatic jaundice.*

2 Through a special change in the blood (hemolysis): *Hematic* (hemolytic) *jaundice.*

3 Through a special kind of intoxication (picric acid): *Picric jaundice*

## I.—HEPATIC JAUNDICE.

Hepatic jaundice, dependent upon retention and reabsorption of bile and biliary pigments, is that which displays in its greatest intensity the well-known symptom-group of jaundice with its *cardinal symptoms, viz*, jaundice of the skin and conjunctivæ and urinary jaundice (from canary yellow to mahogany color, with more or less pronounced decolorization of the stools), and its *associated symptoms* due to bile intoxication, *viz*, slow pulse, itching, loss of weight, depression, oozing of wounds, etc

It should be at once pointed out that this classical symptom-group, which, as we shall see, is of markedly variable origin, exhibits a diminishing degree of intensity of the jaundiced color of the skin, conjunctivæ, and urine in the following three classes of cases.

Maximum intensity: Cholelithiasis, cancer of the pancreas, and chronic obstruction of the bile duct

Intermediate intensity: Catarrhal jaundice, benign infectious jaundice, and picric jaundice.

Minimum intensity: Infectious, cirrhotic, syphilitic, and hemolytic forms of jaundice.

*Hepatic jaundice* may be the result either of an obstruction or impediment to the flow of bile or of disease of the hepatic lobules or dyshepatia.

**Intrinsic (Intracanaliculobiliary) Causes of Obstruction**—**Cholelithiasis**, affecting the gall-bladder, but more particularly the biliary canal or bile-duct, is by far the most important among the possible causes of jaundice; tenderness of the gall-bladder, acute attacks suggestive of hepatic colic, and the history will generally point directly to the diagnosis

**Catarrhal jaundice** comes next, with its usual accompaniment of **febrile gastric disturbance** and running its course in one or two weeks; as a rule the diagnosis of these cases occasions no difficulty.

Exceptionally there have been reported instances of **foreign bodies** that had passed out through the walls of the intestine (fruit stones, grape seeds, and parasitic ascarides or hydatid disease); in *such cases the diagnosis can be made only as an unexpected finding* during an operation or at the autopsy. In the case of a *cicatricial stenosis following duodenal ulcer,* the diagnosis would be made on the basis of the history and the symptoms of duodenal disease.

**Extrinsic (Extracanalicular) Causes of Obstruction**—1. *Outside of the liver:* Usually, **cancer of the head of the pancreas**, which is by far the commonest cause of jaundice of extrahepatic origin. Exceptionally: Secondary tuberculous or malignant glandular involvement at the hilum of the liver, peritoneal bands, adhesions of the biliary channels, lower hepatic surface and colon on the right side, tumors of the kidney, and aneurysm of the abdominal aorta.

2. *Within the liver:* **Cancer of the biliary ducts and liver, hepatic abscess, and cysts of the liver.**

*Disease of the lobules of the liver,* or **dyshepatia**, may result from some intoxication or infection acting injuriously upon the liver cells. It is generally manifest in the lengthy chain of infectious jaundiced states, a mere enumeration of which will here suffice:

*Catarrhal jaundice,* simple or prolonged, always benign, and occurring sporadically and indigenously.

*Benign infectious jaundice,* or pseudocatarrhal infectious icterus.

Pleiochromic jaundice.

Recurring infectious jaundice.

*Grave icterus, usually secondary* to some pre-existing liver disturbance, such as cirrhosis, etc, or to an infectious disease, such as typhoid fever, staphylococcic infection, malaria, etc.; exceptionally as a primary disorder, as in phosphorus poisoning, yellow fever, icterohemorrhagic spirochetosis, etc

This type of jaundice is essentially characterized clinically by a certain symptomatic triad, *viz.*, (1) jaundice, (2) typhoid state, and (3) various sorts of hemorrhage According to the kind of case the condition may be attended with hypothermia, as in colon bacillus infection and phosphorus poisoning, or with fever, as in yellow fever and staphylococcic or streptococcic infection.

Larrey, in his Mémoires, already wrote of an "icteroid typhus" which assailed the troops of the Army of Egypt in 1800 During the War of the Rebellion, over 70,000 American soldiers became afflicted with jaundice. More recently, in Macedonia, the belligerent armies developed many cases of grave malarial bilious fever (intermittent bilious fever, hemorrhagic bilious fever, hemoglobinuric bilious fever, etc ).

Special mention may here be appropriately made of an *apparently primary* variety of *infectious jaundice* which has only of late come to notice. It is manifested in a recurring febrile infectious jaundice, ordinarily accompanied by myalgia and hemorrhage, and brought on by a spirochete discovered and studied in 1913-1915 by two Japanese authors, Inada and Ido, whence the term *icterohemorrhagic spirochetosis* now generally applied to the disease.

This form of infectious jaundice, often a grave disorder, is probably identical with Larrey's icteroid typhus, with the epidemic remittent bilious fever of Laveran, and with the idiopathic grave icterus of Kelsch

It is manifested in a symptom-group characterized by sudden onset, high fever (39-40° C ), a pronounced typhoid state, a progressive jaundice of variable intensity, pains in the extremities, myalgia (especially in the thighs and calves), and joint pains,

albuminuria, and a progressive azotemia which may attain to a very high degree. In one of our fatal cases, the azotemia ultimately rose to 6.80. The provisional diagnosis of spirochetosis can be definitely established only by microscopic examination for the causative germ.

As a matter of fact, icterohemorrhagic spirochetosis may exhibit any grade of severity, from the form suggesting catarrhal jaundice to that representing grave icterus. On the whole one may, however, make out three stages in the course of the disease: (1) A preicteric stage, with predominance of constitutional symptoms ranging from simple diffuse pains to continuous fever suggesting typhoid; (2) an icteric stage, with more or less intense and persistent jaundice and almost constant albuminuria; (3) a stage of slow restoration, with gradual return to normal, or, on the contrary, aggravation of the disease with progressive azotemia and a fatal termination.

A positive diagnosis manifestly requires examination for the spirochete. Its presence is best demonstrated by guinea-pig inoculations. Into one guinea-pig 5 cubic centimeters of blood are injected and into another, 5 cubic centimeters of urine. Where the result is positive, the animal dies in about a week, exhibiting a yellow color of the scleræ, ears, and mucous membranes, and bile pigment, albumin, and spirochetes in large numbers in the urine and in sections of internal organs.

In *hepatic, mechanical jaundice* of the retention type, the bile pigments retained in the blood and passing out in the urine are normal bile pigments (see *Technical procedures*), and the icterus may be said to be *orthopigmentary*.

In *infectious, dyshepatic jaundice*, the result of cell disturbance and pathologic change, the bile pigments retained in the blood and passing out in the urine are normal as well as abnormal bile pigments (see *Technical procedures*), and the icterus may be said to be *metapigmentary*.

## II.—HEMATIC JAUNDICE.

The hematic forms of jaundice, mainly dependent, apparently, upon some disturbance of the blood, or at least accompanied and characterized by such a disturbance, were already clearly discerned

in the eighteenth century. Bianchi's concise reference to the matter (1710), quoted by Boix, leaves no room for doubt in this connection: *"Sunt duo primaria icteri genera: prima classis icterus e vitio hepatis, alterius speciei uteri a causa solutiva sanguinis."* The profound studies of Gubler on hemapheic icterus are well-known to all. The subject has, however, in late years been completely recast, thanks particularly to the labors of the French clinicians Chauffard, Gilbert, Widal, and their followers on the *hemolytic forms of icterus.*.

**Clinical characteristics of hemolytic icterus:**

1. Jaundice generally of slight or intermediate intensity.
2. Coloration of the stools
3. Hemapheic character of the urine (see *Urine*) Absence of true bile pigments, presence of urobilin (acholemic, orthopigmentary jaundice).
4. Absence of the ordinary signs of bile intoxication, *viz.*, absence of slow pulse, itching, xanthelasma, and loss of weight.

**Characteristics referable to the blood:**

1. Anemia.
2. Lowered resistance of the red cells (hemolytic reaction).
3. Granular red cells and autoagglutination of these cells

**Clinical varieties (the least obscure ones) of hemolytic icterus:**

1. Congenital, familial hemolytic jaundice (Gilbert's familial cholemia; Chauffard's congenital icterus of the adult) As a matter of fact, all cases of congenital icterus in the adult should be presumed to be of non-hepatic origin.
2. Acquired hemolytic jaundice Simple jaundice of the newborn, simple post-infectious hemolytic jaundice, hemolytic jaundice of the type of pernicious anemia, and the jaundice of idiopathic paroxysmal hemoglobinuria

### III—PICRIC JAUNDICE.

Picric jaundice results, as the term implies, from picric intoxication, by the ingestion of picric acid It is characterized·

1. By a yellow, icteric hue of the skin, conjunctivæ and urine
2. By coloration of the stools.

3. By the *usual* absence of bile pigments from the urine and blood serum, picric acid being, however, present in these fluids (see *Uranalysis*).

4. By the *usual* absence of the classic signs of bile intoxication, *viz.*, by the absence of slow pulse, itching, and loss of weight.

The procedure described by Castaigne and Desmoulins, which permits the detection of picric acid in the serum of patients with jaundice, is particularly serviceable by reason of its simplicity, reliability, and speed. Fifteen to 20 cubic centimeters of blood are collected by wet cupping or vein puncture and placed in a test-tube An equal volume of a 25 per cent. aqueous solution of trichloracetic acid is added The tube is then closed with the thumb and thoroughly shaken, the mixture poured on an ordinary pleated filter, and the filtrate collected in a well cleaned test-tube.

In the absence of picric acid the filtrate is clear and colorless.

The presence of picric acid is shown by a yellow picric tint of varying intensity upon inspection against a white background.

## IV.—SYPHILITIC JAUNDICE AND JAUNDICE DUE TO ARSPHENAMIN.

The relative frequency of jaundice occurring during arsphenamin treatment has led to the publication of many contributions on the subject which are rather contradictory, some considering the jaundice as a syphilitic jaundice or hepato-recurrence awakened by the treatment and amenable to more intensive medication, while others look upon it as a toxic arsenical jaundice.

In truth, it would seem most extraordinary if the drugs of the arsphenamin series were the only arsenical compounds which might not, in high dosage, act prejudicially on the liver, and certainly nothing is less definitely proven at the present time than that arsenical treatment is indicated and effectual in these cases. *For the present it seems prudent to consider the jaundice which occurs under these conditions as of toxic, arsenical nature, and to interrupt the treatment or replace it by mercurial medication.*

As for chronic jaundice, it should be recalled that Hanot's syndrome and the hemolytic types of jaundice particularly call for a search for acquired syphilis in the former case and for syphilis or inherited syphilis in the latter.

## V.—RELATIVE FREQUENCY OF THE SEVERAL FORMS OF JAUNDICE.

Clinically, the order of frequency of the several varieties of jaundice above mentioned appears to be as follows:

Infectious jaundice, ranging from catarrhal to grave.

Cholelithiasis, simple or with complications.

Hemolytic icterus.

Tumors of the liver

Cirrhosis and syphilis of the liver.

Cancer of the pancreas and biliary tract, etc. (extrahepatic tumors).

Abscess and hydatid cyst.

Picric jaundice

Consideration of the differential diagnosis of jaundice in the subjoined table will be limited to the varieties just given

## JAUNDICE.

| Causes. | History Predisposing or Exciting Causes | Accompanying Local Signs | Accompanying General Signs | Features of the Jaundice | Blood Examination. |
|---|---|---|---|---|---|
| **Infectious jaundice:** *Benign* (type case: catarrhal jaundice). | Various infections, particularly gastro-intestinal. | Generally hepatic congestion. | Moderate and temporary rise of temperature Leucocytosis Slow pulse | **Of intermediate intensity; evanescent.** | |
| *Grave* (type case icterohemorrhagic spirochetosis) | 1. Various infections 2 Pre-existing debility of liver and kidneys 3 Sometimes a primary infection· Spirochetosis | Liver *small* Inconstantly, splenic enlargement | 1 *Typhoid state* 2. Various hemorrhagic phenomena 3 Azotemia 4 Myalgic pains | ***Of intermediate intensity.*** | Leucocytosis. Hyperazotemia. |
| **Cholelithiasis.** | Typhoid fever Obesity, especially in women. Excessive use of cholesterin-yielding *foods* | Hepatic colic Gall-bladder tender, enlarged, and palpable Gall-bladder point of tenderness | Fever wanting or intermittent Slow pulse Itching of skin | Type of **obstructive jaundice** in the presence of a stone in the bile-duct. | 0 |
| **Hemolytic jaundice.** | Familial cholemia. | Liver normal Spleen enlarged | Anemia Asthenia. No itching of skin No indications of insufficiency of the liver Excessive coloration of feces | **Of slight intensity.** Practically continuous subicterus "Hemapheic" jaundice | Abnormal fragility of the red cells. Hemolysis Increase of granular red cells. |

| | | | | | |
|---|---|---|---|---|---|
| Tumor of the liver: *Primary* | ? | Smooth, regular, almond-shaped, progressive *enlargement* of the liver | Anemia<br>Asthenia.<br>"Galloping" emaciation.<br>Rapid cachexia. | Of intermediate intensity. | Anemia of the "pernicious" type. |
| *Secondary:* | Pre-existing cancer of stomach or intestine | *Nodular,* irregular, progressive enlargement of the liver | | | |
| Cirrhosis and syphilis of the liver. | 1 Alcoholism<br>2 Syphilis. | 1 Small liver (atrophic cirrhosis) or enlarged liver (biliary cirrhosis) or hob-nail liver (syphilitic)<br>2 High portal pressure<br>3 Enlarged spleen | Anemia<br>Emaciation. | Subicterus, or slight, ambiguous jaundice | 1 Variable<br>2 Positive Wassermann. |
| Extrahepatic tumors (pancreas, bile-ducts, or duodenum). | Second half of life | 1 Evidences of the tumor<br>2 Dilated gall-bladder | Anemia<br>"Galloping" emaciation<br>Itching of skin. | Type of obstructive jaundice, intense and *progressive* | Anemia |
| Abscess of the liver. | Dysentery<br>Malaria | Liver enlarged, tender, with pre-eminently localized pain | Continuous and intermittent fever<br>Chills.<br>Leucocytosis. | Of intermediate intensity. | Leucocytosis |
| Picric jaundice. | Suspected malingering | *0* | *0* | Subicterus. | Presence of picric acid in the blood serum. |

JOINT PAINS.
ARTHRALGIA.
RHEUMATISM.

[*ῥευματισμός, from ῥεῦμα, flux. Rheumatism.*]

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Pains in the joints (arthralgia) occur so often, in such a variety of situations, and in so many forms that an analytic and synthetic presentation of the subject would seem to be almost an impracticable task, particularly since it brings up the question, as yet obscure in many respects, of "rheumatism." Hence the author's belief, departing from the plan followed in other chapters of this work, that it is best here not to analyze the "innumerable and protean joint disturbances," but to arrange them in logical groupings from the standpoint of pathologic physiology and to recall the general clinical rules which permit of the making of a concrete diagnosis in these cases

To give an idea of the frequency with which the term "rheumatism" is improperly applied to the most varying clinical conditions, one need merely recall the figures published by Deaderick, analyzing 100 cases labelled "rheumatism" and detecting 53 mistaken diagnoses which were grouped thus

Eighteen cases of syphilis with positive Wassermann reaction, 8 cases of neuritis, 4 cases of tuberculosis, 4 of flat foot, 2 of neurasthenia, 2 of arteriosclerosis, 2 of tabes dorsalis, 1 of chronic nephritis, 1 of chronic gastritis, 1 of progressive muscular atrophy, 1 of malaria, 1 of pernicious anemia, and 1 of myelitis.

*Joint Pains* —Rheumatism.—Whereas practically definite pathologic and clinical concepts are expressed in the terms diabetes, gout, and obesity, this is far from being the case with the term rheumatism Usage has given to this word a vague, inaccurate, extremely defective meaning because it is far too comprehensive; this term we shall accept, however, without attempting useless explanations of it, the essentially practical aim of the work making it necessary to exclude all hair-splitting and fruitless terminologic discussions

Medical men are constantly speaking, then, of acute articular rheumatism, of gonorrheal rheumatism, of tuberculous rheumatism, of gouty rheumatism, of deforming rheumatism, of trophoneurotic rheumatism, of muscular rheumatism, etc. There is no doubt that it would be much better to use in most instances strictly concrete and determinate pathologic terms such as arthritis, osteoarthritis, neuralgia, myalgia, etc, following them with special qualifying words such as gouty, gonococcic, tuberculous, saturnine, traumatic, etc, and to make of the qualifying term *rheumatic* itself a specific designation applying to certain relatively well defined clinical entities such as acute, frank, rheumatic multiple arthritis, or progressive, deforming, rheumatic multiple arthritis Yet, we repeat, nosologic usage has already settled the matter otherwise and has given to the term *rheumatism the very vague signification of a disorder characterized especially by pains in the joints;* we shall accept it thus, faulty as it is—and all the more willingly since in practical work the often difficult question of diagnosis between most varied sorts of joint disturbance, infectious and metabolic, is continually arising and it seems useful and eminently practical to attempt a comprehensive classification and general review of the clinical features of the joint disorders most commonly encountered

To find and keep to the proper path in the maze of joint disturbances, it is necessary to refer back to the etiology whenever the pathogenic cause of the condition can be determined This is, of course, not always, nor even often, the case, hence the need of accepting, in lieu of a better one, the following hybrid classification, based partly on the etiology and partly on the clinical features

I. Acute joint disorders.

A. *Acute articular rheumatism.*

B *Infectious pseudorheumatism, or better, infectious arthritis (ordinary joint infections, or specific, toxic-infectious disorders)*:

gonorrheal,
tuberculous,
syphilitic,
scarlatinal,
polymicrobic,
miscellaneous (post-influenzal, post-pneumonic, puerperal, post-anginal, post-typhoid, etc.).

C. *Acute gouty arthritis.*

II. **Chronic arthritides.**

A. *Chronic gouty arthritides.*

B. *Generalized chronic rheumatism.*

(*a*) With erratic manifestations (articular, muscular, neuralgic, etc.).

(*b*) With local manifestations (Heberden's nodes, camptodactylia, drumstick fingers, rhizomelic spondylosis, etc.).

(*c*) Progressive deforming rheumatic polyarthritis (nodular rheumatism).

C. *Arthritis deformans, mono- or oligo- articular.*

III. **Trophoneurotic.—Neurotrophic.**

(*a*) Joint disorders following neuritus (zona), myelitis (tabes), encephalopathy (hemiplegia), etc.

(*b*) Amyotrophic arthropathies due to spinal lesion, the latter either primary or secondary to the reaction of the joint inflammation upon the neuraxis.

(*c*) Generalized dystrophy.

IV. **Traumatic.**

(Sprains, fractures near joints, wounds, and foreign bodies.)

The latter group is of interest in this connection only when the traumatism results in local infectious or nutritional disturbance.

The above simple classification is of real practical service; it is sufficiently clinical in type and seems deserving of recommendation provided its somewhat restricted scientific basis is always kept in mind In other words, clinical use of this tabular scheme necessitates familiarity with the three following clinical laws:

I. **There is not necessarily any relationship between the cause of a joint disturbance and its clinical modality.**—Thus, gonorrheal joint disease may occur in any of the following forms:

Acute febrile arthritis (gonorrheal rheumatism).
Suppurative arthritis.
Plastic, fibrous, ankylosing polyarthritis.

Again, tuberculous joint disease may occur as:

Acute febrile arthritis (tuberculous rheumatism).

Subacute serous arthritis (hydrarthrosis).
Suppurative osteoarthritis (white swelling).
Fibrous, ankylosing osteoarthritis.

Even the term tuberculous arthritism has been used by some. Consequently:

**II. A given clinical type of joint disturbance may result from different pathogenic causes.**—Thus, *progressive deforming polyarthritis* may be the end-result of acute rheumatism, of gonococcic infection, of tubercle bacillus infection, and, even more frequently, of as yet imperfectly defined causes, among which are mentioned preeminently exposure to cold and dampness and, as a subsidiary factor, thyroid insufficiency.

Again, *acute exudative febrile arthritis* may be brought on, ordinarily by acute rheumatism, rather frequently by gonococcic infection, exceptionally by tubercle infection, etc.

**III. There is no definite line of demarcation between the three types of joint disturbance (toxic-infectious, dyscrasic, and neurotrophic).**—Or at least, if a few perfectly definite clinical species do exist, such as acute rheumatism, the gouty arthropathies, and the tabetic arthropathies, most of the above mentioned clinical species, of obscure and diverse etiology, do not constitute definite clinical identities, but mere syndromes which may be brought on by different pathogenic causes [toxic-infectious, humoral (exogenous and endogenous), or neurotrophic].

Further, it is readily conceived that some toxic-infectious disorder, reacting upon the functions of the endocrin glands, for example, or upon the neurotrophic cells, might actually bring about the humoral or trophoneurotic degenerative changes which are recognized or suspected as being at the bottom of the majority of the so-called diathetic chronic joint disorders—the various forms of chronic rheumatism

As in the case of diabetes or obesity, one is led, then, to the conception of *chronic rheumatism as a trophoneurotic clinical syndrome affecting particularly the joints, of toxic-infectious or dyscrasic origin, these two pathogenic factors being present either separately, in combination, or as a subordinate cause*

On the whole, the joint tissues react, whatever pathogenic agent they may be subjected to, in but a few ways, *viz*, pain,

congestion, inflammation, serous exudation, suppuration, and fibrosis.

Any joint disorder, of whatever cause (traumatic, infectious, dyscrasic, or nervous) may pass through three distinct stages: Acute stage, chronic stage, and stage of deformity.

Some forms stop in the acute stage, as is generally the case in rheumatic fever; others in the chronic stage, as is frequently the case in tuberculous arthritis; a few begin in an acute stage and terminate in the deforming stage, as is frequently the case in gonorrheal joint disease; certain disturbances, moreover, cause deformity from the start, as in nodular rheumatism. All these varieties are commonly met with

The disease may attack from the start or in succession the synovial membrane, the periarticular aponeuroses, the muscles and tendons in the vicinity of the joint, the periarticular bony surfaces, the nerves, and the marrow; all the tissues, in fact, including the skin, cellular tissues, and vessels, may undergo trophoneurotic degeneration—a matter of common observation in chronic forms of rheumatism

It seems not inappropriate here to present an excerpt from the communication of P. Le Gendre to the Académie de médecine on May 9, 1911, regarding the pathogenesis and prophylaxis of the so-called "rheumatic" affections:

To explain the origin of the rheumatic disorders and their recrudescences, it is necessary to bring in two parallel series of factors, *viz*, (1) the intoxications and toxic-infectious states, and (2) faulty hygiene of the locomotor apparatus

The latter might be said to be the initial and primary factor, producing a favorable soil for the activities of the former; it would thus underlie the entire clinical history of rheumatism in all its forms, both those set apart under the term pseudorheumatism, and the gradually decreasing number which are still considered instances of true rheumatism.

In the case of the latter, Bouchard, through his studies of their concurrence with other morbid conditions, brought out their *relationship with the diseases due to slowing of the nutritive process*, such as obesity, diabetes, gout, migraine, calculous disorders, asthma, etc., and with the so-called arthritic diseases. This implies that they

are likewise dependent upon arthritism—the bradytrophic diathesis of Landouzy, or dystrophic diathesis, if one uses the term proposed by Fernet. This relationship seems also to be shown by clinical observation of the past histories of the patients themselves and their family histories.

Might one not conceive of this relationship, however, as being the *result of the influence of faulty hygiene of the locomotor apparatus upon general nutrition?*

It is impossible that an apparatus of such importance in the living system should not play a very prominent rôle in the activity of interstitial metabolism. In it, along with the digestive tract and the nervous system, are to be found the three great sources of nutritive disturbances—acting through different mechanisms but frequently in combination.

A *diet which is defective through excess or improper selection of foods,* imperfect elaboration of the latter owing to impaired digestive functions, autointoxication by poisons of gastrointestinal origin resulting from prolonged stasis of the digestive residue in one or another portion of the alimentary tract (dilatation of the stomach, dyspepsia with ileocecal stasis, and coprostasis either of liquid or solid matters in the colon), and defective functioning of the liver—these, doubtless, are the causes of arthritism.

An *excessive, disturbed functioning of the nervous system,* inhibiting tissue metabolism or secretion by the endocrin glands which supply the ferments indispensable for such metabolism, should also be taken into account in the production of certain arthritic diseases, and it has been necessary to appeal to it in the pathogenesis of obesity, of diabetes, and of gout, according as the metabolic processes concerned were those of fat, sugar, or uric acid production, or fat, sugar, or uric acid destruction.

As a counterpart to these factors, we shall accept the view that there occurs also a group of disorders characterized by slowed nutrition which is dependent upon a *primary disturbance of the locomotor apparatus* and that *it is this disturbance which is at the bottom of the so-called true rheumatic disorders*—predisposing the various constituent parts of this apparatus to become unduly sensitive to cosmic factors as well as to endogenous toxic influences, and

to react against them by such manifestations as pain, hyperemia, exudation, or proliferation.

Perhaps this dystrophic state of the serous, fibro-connective, osteo-cartilaginous, and muscular systems also predisposes them to being more easily injured by exogenous bacterial or toxic attacks and to reacting against the latter by various local changes which may progress to the point of actual suppuration

Among the characteristic effects of the bradytrophic diathesis is an excessive sensitiveness of the vasomotor system; as Cazalis and Sénac have stated, it is a congestive diathesis, which favors hyperemia, edema, and oversecretion, it also exhibits an excessive tendency to painful manifestations or algias. Thus, it carries with it a tendency to react excessively to cosmic factors in the production of congestion, edema, and exudation in the serous membranes of the joints and the synovial coverings, and to translate into pain all functional disturbances of the various constituent parts of the locomotor apparatus.

To recapitulate, the motor apparatus contributes in two respects to the general functioning of the system

Through its serous membranes, connective and fibrous tissues, and marrow tissue, *it constitutes a part of the connective tissue,* lymphatic, and leucocytic *defensive system;* it serves as a locus for the unloading and destruction of bacterial agents and the soluble, organic or mineral poisons of bacteria.

By the functioning of its masses of muscle tissue, *it takes part in the processes of nutrition;* it uses up glycogen and forms lactic acid and many other products of disintegration

*From the first of these standpoints,* it is destined to be the seat of infectious rheumatic disorders, or *pseudorheumatism.*

*From the second standpoint,* it may contribute to *the production of the bradytrophic diathesis,* and when it itself takes the consequences of the latter, becomes the seat of what is still termed *true rheumatism,* with the associated extraordinary sensitiveness to cosmic influences

Always at the bottom of the condition, however, is faulty hygiene of the motor apparatus.

This pathogenetic conception is perhaps not actually a new theory; it is, at least, a more comprehensive interpretation of

known facts and current views, capable of serving as a link between the older theories which are only apparently in disaccord—it is thus a common meeting-ground for purposes of conciliation.

It offers the particular advantage of serving as a basis for therapeutic, and especially prophylactic, indications.

In the prophylaxis of the arthritic dystrophy and of the nutritive diseases—apart from the regulations concerning dietetic hygiene, so much emphasized, and justifiably so, in modern contributions, and apart from the hygiene of the nervous system, which has been rather more neglected—highly important indications appear to the author to be as follows:

1. One should *regulate the hygiene of the motor apparatus* with the greatest care, *beginning in early childhood,* in all children, but more especially those the offspring of rheumatic individuals. There should be *sufficient,* but *never excessive*—particularly regular—*daily exercise* of all the motor structures

2. One should place these structures in a state of defence against cosmic agencies, particularly exposure to cold, by *a systematic, progressive training toward tolerance of cold, by stimulation of the skin functions* Instead of defending one's self passively against cosmic influences, it is better to activate the play of the vasomotor reflexes and skin excretions by dry rubbing or rubbing with alcohol, cold affusions, and hydrotherapy of the "hardening" type

Again, when the rheumatic tendency, *i.e.*, the state of lowered resistance of the motor apparatus to cosmic agencies and endogenous onslaughts, has actually shown itself, one should *look for and overcome,* insofar as is possible, any *sources of infection and intoxication* which the body may be harboring These are detected by a careful clinical study of the functional activities of the digestive tract and the annexed glandular organs, the nasopharyngeal cavities, the genital organs, and the endocrin glands, and by analysis of the blood and urine. Though there are rheumatic subjects with excessive uric, lactic, or oxalic acidity, there appear also to be some with lowered acidity

Sources of intoxication should be removed by the use of the best procedures now known; the emunctories should be assisted, and insofar as is practicable, the faulty chemical state of the body fluids should be corrected.

All this can be done without neglecting the use of remedies that may allay the rheumatic symptoms or influence the pathologic results of the disorder—measures selected from the pharmaceutic realm and more especially from physical therapy.

**It is well to emphasize again the marked importance of looking for some chronic or subacute focus of infection in the presence of cryptogenic joint affections.** The nasopharynx and teeth (caries or infectious processes) should, in particular, be investigated. *In the course of a study of 1000 cases of acute joint* disturbance, more or less closely suggesting acute rheumatism, Lambert (*Jour. Amer. Med. Assoc.*, Apr. 10, 1920) found 243 instances of inflammation of the nasopharynx and 683 of dental infection. Such infections are of frequent occurrence in chronic *joint disorders. Indeed, the author has personally seen some* cases of subacute or chronic joint involvement clear up after appropriate treatment of the teeth and nasopharynx had been given.

Attention may also be directed to the relative frequency, formerly overlooked, **of syphilis as the cause of certain cases of polyarticular, deforming rheumatism** of the type of arthritis sicca or subacute arthritis running a progressive course, with deformation in and about the joints, resulting in segmental changes in relation to the affected joints, muscular atrophy, exacerbations of synovial distention or hydrarthrosis, and arthralgic or neuralgic pains (H. Dufour, Attinger, etc.)

Aside from the *gummatous joint involvements*, which are *relatively easy to diagnose*, whether in the earlier stage with hard, painless, rounded nodes and *almost complete absence of joint reaction*, or the stage of softening, with rounded, punched out, nearly painless ulcerations, recognition of the other joint disturbances is more difficult. "It is by clinical means alone (anamnesis, significant stigmata, serum tests) that one must endeavor to elucidate, in particular as to its relations to lues, the causal diagnosis, often so obscure, of hydrarthrosis of the knee" (Broca, *Presse méd.*, Nov. 2, 1921).

## LOSS OF WEIGHT.

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The body weight is a factor of considerable clinical importance. The scales are to be thought of as an essential piece of medical equipment, as necessary as the clinical thermometer. Along with the blood-pressure instrument, they constitute the

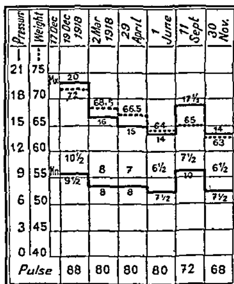



Fig 806—Case 2500 F., 1860, 154 cm Loss of weight.

thermometer of chronic cases, which will detect many instances of as yet latent functional failure, and permit of accurately tracing the course of many morbid states

Experience has shown that for each individual there is a normal weight characteristic of health, which, in the event of perfect functional equilibrium, ranges only between rather restricted limits. The author's normal weight, as recorded for nearly thirty years, has always ranged between 74 and 75 kilograms. Below 73, there appear unquestionable evidences of physical and mental weakness (such a test was made ten times in the course of re-

peated reduction cures); above 76, manifestations of plethora develop.

Theoretic *expressions of this physiologic body weight have* been vouchsafed, the simplest of which consists in the assumption that the normal weight is equal in kilograms to the number

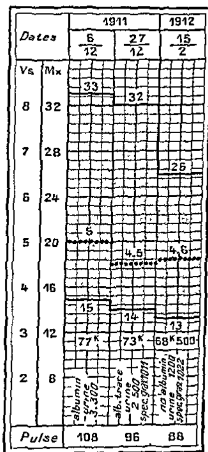



Fig 807—Case 252, 49 years, 160 cm Gradual retrogression in a cardiorenal case with good compensation.

of centimeters of height above one meter. Thus, for a person measuring 170 centimeters it would be 70 kilograms, and for one measuring 154 centimeters, 54 kilograms, etc.[1]

Observation shows that this rule is correct only for medio-

[1] One hundred centimeters = 39.37 inches; one kilogram = 2.2046 pounds Avoirdupois.

linear individuals, of intermediate morphologic type, *i.e*, persons the ratio of whose height to the biaxillary diameter is in the neighborhood of 5.8 (5.6 to 6), as in the subject referred to below:

(Case 3049). *Mediolinear,* $\frac{170\text{ cm}}{29\text{ cm}}$ (5.85), whose normal weight is 68 kilograms.

In a brevilinear or stocky individual, with a broad biaxillary diameter and in whom the ratio referred to is below 5.6, the

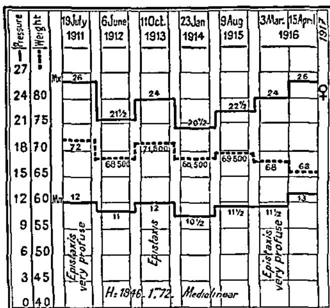



Fig 808.—Case 467 Cardio-arterio-renal sclerosis

normal weight generally exceeds by a few kilograms the figure obtained by the preceding calculation, as in the following subject:

(Case 399) *Brevilinear,* $\frac{170\text{ cm}}{31\text{ cm}}$ (5.44), whose normal weight is 74 kilograms

In a longilinear individual, on the other hand, the preceding ratio being above 6, observation of subjects in a state of perfect nutritive equilibrium yields lower figures, as in the following case

(Case 3207) *Longilinear,* $\frac{180\text{ cm}}{29\text{ cm}}$ (6.20), whose normal weight is 73 kilograms

These three concrete, not theoretic, examples point clearly to the necessity of taking into account at least the vertical and transverse

measurements in calculating *theoretically* the normal weight of an individual.

The theoretic normal weight of a subject who is:

Mediolinear, *is expressed approximately in kilograms by the number of centimeters by which his height exceeds one meter.*

Mediolinear: *Height, 170 centimeters = weight, 70 kilograms.*

Brevilinear: *May exceed the above figure, the correction sometimes reaching one-tenth of the whole.*

Brevilinear: *Height, 170 centimeters = weight, 70 to 77* $(70 + \frac{70}{10})$ *kilograms.*

Longilinear: *Should be lower than the figure in mediolinear subjects, the correction sometimes reaching one-tenth of the whole.*

Longilinear *Height, 170 centimeters = weight, 70 to 63* $(70 - \frac{70}{10})$ *kilograms.*

* * *

Even with due corrections, however, such theoretic, approximate results hold good only in subjects who are nearly normal; continued concrete observation without preconceived notions leads to the conclusion that in individuals with inherited constitutional defects the foregoing rules do not hold good. There are some **constitutionally lean** persons whose normal weight in a state of physiologic equilibrium may be much below the theoretic figures previously mentioned; again, there are **constitutionally stout (obese)** persons whose normal weight may be decidedly above these figures. One of the author's patients (Case 2510), an adult (born in 1885), an unusually longilinear subject with inherited dystrophy ($\frac{183 \text{ cm}}{26 \text{ cm}}$ Index: 7 05[1]) was observed to vary from 52.8 to 61 kilograms He could never be brought to weigh more than 61 kilograms. This is, of course, a subject suffering from constitutional hyposphyxia, asthenia, varicose vessels, etc., with a diabetic family history.

In this connection one might discuss the ethnic, familial, and pathologic factors resulting in the development of special types of constitution; such a discussion, however, would lead us too far afield.

Loss of weight may thus constitute a favorable process, and one which the therapeutist will seek to obtain in many instances where the patient's weight is above normal and this condition is accompanied by pronounced disturbances such as plethora and evidences of cardiovascular inadequacy. (See *Plethora* )

At this point mention may be made of the remarkable relationship generally existing between the body weight and the blood-pressure. In a high pressure case (plethoric or cardiorenal) undergoing improvement, the weight and blood-pressure go hand in hand (Figs 806 and 807). In a low pressure case (anemia, asthenia, and certain forms of hyposphyxia), the weight and blood-pressure rise simultaneously This is a point both of prognostic significance and of actual practical import.

In the course of certain cases of arteriosclerosis the correspondence of the weight and pressure is most striking, and where this correspondence is observed to cease, the weight falling while the blood-pressure is rising, certain aggravation of the sclerotic process is indicated and the prognosis rendered less favorable. This condition was noted in the case represented in Fig 808, which was under observation for a prolonged period

In short, *loss of weight generally goes hand in hand with diminution of blood-pressure, and gain in weight with elevation of blood-pressure (at least in well compensated subjects) Lack of accord in the progression of these two factors is of marked prognostic significance.*

* * *

Loss of weight is a very common clinical event, the cause of which should be inquired into with great care. Its import is sometimes obvious, as, for example, in the case of an individual whose work has increased while the amount of food taken has diminished. The same is likewise frequently true where the loss of weight is accompanied by manifest signs of some disease, such as tuberculosis, typhoid fever, acute infections, gastric disorders, enteritis, etc But much oftener still, a careful inquiry to ascertain the cause is required

The author has made note of the various clinical cases which came to him complaining chiefly of loss of weight which had

excited their apprehension. These cases may be classified in the following groups, given in the order of frequency:

1. **Gastrointestinal disorders,** and more particularly the frequent and common forms of *atonic (hyposthenic) gastrointestinal dyspepsia* and *acute or chronic enteritis.* The main diagnostic features connected with this group of cases will be found in the section on *Dyspepsia.*

2. **Infections,** especially those of **tuberculous** and **influenzal** nature, and secondarily all *acute or chronic infections* (choleroid states and typhoid fever at one extreme and syphilis at the other). The sequence of events in these cases is too obvious to require further comment than the following, to wit: Where the occasion presents, one should examine systematically for the characteristic evidences of the infections (auscultatory signs, sputum examination, leucocyte count, temperature curve, etc.), and carefully avoid making the least premature statement involving the serious risk of a mistaken diagnosis

3. **Depressive psychoneuroses,** together with **exophthalmic goiter and Addison's disease.** During the course of the year 1918, a large number of persons sought the author's advice on account of *loss of weight,* in some instances very marked, *e g*, 8 to 12 kilograms (20 to 30 pounds) or more. In a few cases food restrictions, particularly that referring to bread, seemed to be the main cause. In the greatest number of instances, the loss of weight was accompanied by numerous evidences of general depression (insomnia, weakness, exaggerated emotivity, pessimism, despairing thoughts, "cafard," etc.). Examination of the body itself yielded practically negative results, revealing, however, a *reduction of blood-pressure* which was often very pronounced The anxiety and worries undergone in that trying year were unquestionably the exciting cause of this morbid condition, which the author is disposed to term *depressive psychoneurosis with impaired nutrition and low blood-pressure.* All these patients recovered under the threefold influence of tonic medication, optimistic psychotherapy, and the final triumphant victory of the Allied cause.

*Exophthalmic goiter* is sometimes the source of a most striking loss of weight, amounting practically to cachexia. The same is true of *Addison's disease*

4. Comparable with the foregoing types of cases are *certain* instances of *loss of weight* accompanied by temporary glycosuria in overworked individuals, especially intellectual workers. The author has seen a number of these cases among his colleagues in the course of the last few years.

Such a loss of weight may, however, as is well known, be an initial indication of *diabetes,* particularly the grave cases of pancreatic diabetes, but also sometimes in the so-called neuroarthritic, hepatic, and nervous forms of diabetes.

**Practical conclusion: Never omit uranalysis in a case of loss of weight.**

5. **Tumors,** in whatever situation, though more especially tumors of the digestive tract, constitute the most dangerous factor of loss of weight. Tumor should always be thought of in an elderly person who begins to lose weight and whose general condition exhibits continuous and inexorable deterioration. At the least suspicion of trouble, a systematic examination must be carried out, including inquiry for occult bleeding from the alimentary canal through examination of the stools, careful examination of this canal by fluoroscopy after ingestion of a bismuth meal, etc., and will very often settle the question. Pyloric stenosis, cancer of the large bowel and rectum, and cancer of the liver are by far the commonest medical tumors, the possibility of their presence should always be borne in mind, and in particular, one should never forget to palpate the rectum in all suspected cases

6 **Arteriosclerosis** is also a common cause of impaired nutrition The age of the patient, the high blood-pressure generally existing, and the coexisting signs, generally multiple (tortuous temporal vessels, signs elicited upon auscultation over the aorta, nycturia, etc.), without taking into account the state of hydremia frequently present and manifesting itself in lowered viscosity of the blood, will insure the correct diagnosis

7. Again, mention may be made, as regards more exceptional cases, of **certain intoxications,** *viz*, **alcohol** in large amounts, through the resulting gastrointestinal dyspepsia and hepatic cirrhosis, and **morphine** in chronic poisoning, both of which conditions generally cause loss of weight

8. Lastly, mention may be made, as a species of clinical curiosity, of a condition which, while rather common in an attenuated form in women, is very uncommon in its well-marked form, *viz.*, **progressive lipodystrophy** (BARRAQUER-SIMONS'S DISEASE), characterized by a gradual and almost complete disappearance of adipose tissue and subcutaneous cellular tissue from the upper portions of the body, combined with adiposity of the parts from the umbilicus down. In its attenuated form it sometimes leads to a singular conformation which might be described thus: Venus de Milo above the umbilicus and the Hottentot Venus below it. In its pronounced form the face, chest, and abdomen present a skeletal aspect, while the more dependent parts become so overburdened with fat as to suggest elephantiasis Whether this condition is of endocrin or nervous origin has not as yet been ascertained.

* * *

In brief, in cases of **marked and persistent loss of weight**, a comprehensive examination is, as always, indicated, the essential procedures of examination, however, the various combinations of which with the factor, loss of weight, will permit of definitely grouping 95 per cent. of the cases are the following:

(*a*) **Blood-pressure determinations.**
(*b*) **The temperature curve.**
(*c*) **Uranalysis** (amount, specific gravity, sugar, and albumin).
(*d*) **Careful examination of the digestive tract.**

# LOW BLOOD-PRESSURE.

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Inclusion in this work of a short section on **low blood-pressure** as a counterpart to that on high blood-pressure already presented is warranted 1. Because this condition is very frequently met with 2. Because it is often one of marked clinical significance. 3. Because among chronic cases, at least, determination of the blood-pressure now appears to the author just as essential as that of the temperature or pulse in acute cases.

In this section consideration of the subject of low blood-pressure will be limited to a short discussion of the symptomatic import of low systolic, diastolic, and pulse pressures, the reader being referred for the definition of the terms systolic, diastolic, and pulse pressure to the section on *High blood-pressure.*

**Low systolic pressure** may be put down as commencing at 120 to 130 millimeters of mercury, as determined with the Pachon blood-pressure instrument [The readings with this instrument are somewhat higher than those afforded by the ordinary mercury sphygmomanometer.—TRANSLATOR ] The lowest readings the author has met with in adults were 70 and 80 millimeters.

A *continuous low systolic pressure* is found almost exclusively in *anemias,* in the *tuberculous,* in *neurasthenics,* in *adrenal insufficiency* (Addison's disease, post-infectious adrenalitis, etc ), and in a rather important group of cases not specifically grouped until lately, *viz*, cases of low blood-pressure through debility of the cardio-arterial functions, congenital or familial, perpetuated or made worse by a mode of life excluding all physical exercise—a condition the author has described under the term *hyposphyxia.*

Low blood-pressure is one of the almost constant attributes of the *disorders causing cachexia, viz*, tuberculosis, cancer, and defective nutrition; a progressive and inveterate low blood-pressure is generally a forerunner of death.

A *temporary reduction of systolic pressure* may be observed in the course of infectious diseases and during convalescence from them, as well as after hemorrhage or operative shock. The prognostic importance of low pressure in the last-named class of cases is well known; in thoraco-abdominal wounds, progressive reduction of blood-pressure points to a continuing hemorrhage.

*Traumatic shock* is a general somatic state met with after wounds and characterized by a pronounced and persistent deficiency of blood-pressure, with frequent pulse, pallor, sweating, shallow and frequent respiration, and extreme weakness with a tendency to syncope

**Low diastolic pressure**, 80 millimeters or below (oscillometric or auscultatory methods), is met with only in conjunction with the low systolic pressure of anemic and cachectic cases and in *aortic regurgitation*. The lowest reading obtained by the author in an adult was 50 millimeters, in a case of pernicious anemia. Low diastolic pressure is of diagnostic import only in aortic regurgitation, but in this condition its significance is very clear-cut and may be expressed as a definite rule: *The combination of low diastolic pressure* (80 *mm. or below*) *with high systolic pressure* (170 *mm. or above*) *is pathognomonic of aortic regurgitation.*

As for the significance of the **pulse or differential pressure**, following are certain justifiable statements on this subject: The pulse pressure is *in a measure* a sphygmomanometric reflection or expression of cardiac power *Persons with constitutional debility* and congenitally small hearts exhibit a low pulse pressure (20 to 40 mm), while well compensated arteriosclerotic cases with hypertrophied "ox hearts" show a very high pulse pressure (100 to 250 mm.). In cases of cardiac insufficiency with low pulse pressure the *pulse pressure rises in proportion as* the heart regains its functional power and diuresis takes place, etc.

In some cases of *"shock,"* the pulse pressure may be completely obliterated. In these cases a fatal termination has, so far, always occurred. The prognosis of "shock" is the less favorable in proportion as the pulse pressure sinks lower and *the oscillometric index* falls more rapidly.

Three points relating to low blood-pressure seem to require further discussion from the practical standpoint, *viz.:*

1. Low blood-pressure in tuberculosis
2. Low blood-pressure in adrenal insufficiency.
3. Low blood-pressure in hyposphyxia.

1. Low pressure is so constantly present in tuberculous cases that: (1) It may be considered an evidence of tuberculosis to the same degree as cough, loss of weight, fever, anorexia, lassitude, etc ; taken alone, it is of very little symptomatic value, but in conjunction with the preceding manifestations it assumes marked significance. (2) The finding of a normal or increased pressure warrants the conclusion that either tuberculosis is absent or some complication, generally renal, is present; all the tuberculous subjects in whom the author found high blood-pressure were also suffering from nephritis

Nevertheless—and this statement applies whenever one is dealing with low pressure—one must always take into account the *hypertensive reaction of nervous origin* which inevitably occurs at the first blood-pressure examination Whence the following admonitions·

(1) At the first examination, one should make two successive pressure determinations at a few minutes' interval [The discrepancy, often pronounced, between the first and second readings (the latter always being lower) affords a good index of the emotivity of the patient concerned ] (2) Further blood-pressure determinations should be made at later examinations The emotional and accidental, temporary, hypertensive factor is thus eliminated, and the morbid, essential, and permanent factor alone kept in view.

2. Low blood-pressure is one of the three permanent evidences of adrenal insufficiency (hypoadrenia) One may state that the symptom-group *low pressure, asthenia,* and *Sergent's white line* is pathognomonic of adrenal insufficiency, the extremely frequent clinical occurrence of which has been discovered through recent investigations. In its highest expression this condition is manifested by the well-known classic syndrome of *Addison's disease,* long since ascribed to degenerative changes in the adrenal glands and accompanied by *characteristic pigment changes in the skin and mucous membranes.* The conclusion from the recent investiga-

A *temporary reduction of systolic pressure* may be observed in the course of infectious diseases and during convalescence from them, as well as after hemorrhage or operative shock. The prognostic importance of low pressure in the last-named class of cases is well known; in thoraco-abdominal wounds, progressive reduction of blood-pressure points to a continuing hemorrhage

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Three points relating to low blood-pressure seem to require further discussion from the practical standpoint, *viz.:*

The first type exhibits a pulse pressure which is high in comparison to the normal or low blood viscosity; these are hypersystolic, hypersphyxic cases, the permanent hypersphyxia being represented by arteriorenal sclerosis.

The second type, which is the subject of the following brief study, shows, on the other hand, a viscosity which is high as compared with a normal or low pulse pressure. These are hyposphyxic cases.

**The hyposphyxic syndrome.**—Hyposphyxia consists of the combination of an absolutely or relatively low pulse pressure with a high blood viscosity. These two factors, simultaneously present, constitute the highest expression of the condition of *sluggish circulation so frequently noted in young girls, sedentary individuals, pretuberculous subjects,* etc., and characterized especially by *weak pulse,* a *livid skin surface, habitual coldness and cyanosis of the extremities,* a *tendency to venous plethora, varicose veins, enlargement of the liver, unusual sensitiveness to cold,* etc.

Hyposphyxia is almost constantly associated with *pluriglandular insufficiency,* of which it is a dominant feature and upon which it depends. In hyposphyxics there are noted, indeed, gastrointestinal dyspepsia due to inadequacy of the several digestive glands, various disturbances long since attributed to insufficiency of the endocrin glands (thyroid, ovaries, adrenals, pituitary, etc.), *viz.,* headache, migraine, dysmenorrhea, asthenia, asthma, disturbed nutrition of the hair, etc.

The hyposphyxic syndrome is specifically mentioned in many descriptions of the syndromes due to insufficiency of glandular functions.

Stress is to be laid on the fact that *hyposphyxia* is only a symptom-group and not a definite disease entity, and that one may distinguish organic and functional forms of hyposphyxia, as well as constitutional inherited, and accidental (*e.g.,* post-infectious) forms. Precisely the same is true of the syndrome of pluriglandular insufficiency.

This ascendancy of the circulatory factor over the neuro-musculo-trophic factor had already been clearly expressed by Brissaud in relation to feeble children (Bauer) and to mitral dwarfism. "As soon as a certain degree of narrowness of the arteries

tions referred to seems to be that most instances of infectious, post-infectious, or cachectic low blood-pressure are wholly or partly the result of adrenal insufficiency, whether there exists an actual adrenalitis or merely a temporary insufficiency (Loeper). *This interpretation, however, is still a moot point.*

3. **Hyposphyxia** is a circulatory symptom-group a short discussion of which seems warranted in view of the fact that it has been known and described only in relatively recent years.

*Blood-pressure and blood viscosity*—The author has shown in the course of the last few years that in subjects whose cardiovascular system is well balanced and at no point impaired there exists a rather close relationship between the differential or pulse pressure, an expression of the power of the heart beat, and the viscosity of the blood, an expression of the resistance opposed by the blood to the circulation. With a low viscosity, as in anemics, there corresponds a low pressure, with a moderate viscosity, as in normal subjects, a moderate pressure, and with a high viscosity, as in plethoric, full-blooded persons, a high pressure. *In short, in the person who is normal from the cardiovascular, or better the circulatory standpoint, the pressure goes hand in hand with the viscosity.* The converse, however, is not always true, for reasons set forth at length in an earlier work of the author's[1] devoted to a study of this question

Furthermore, this relationship, which the author was enabled to demonstrate only after extensive observations, will seem obvious to any one who will call to mind the fact that the energy required to cause a fluid to circulate in a given canal system is proportionate to the resistance offered by the fluid, *viz.*, to its viscosity.

Such is the natural relationship of the pulse pressure and blood viscosity.

Clinical observation leads to the detection of two radically opposed abnormal sphygmoviscosimetric types of cases in which there is disharmony between the pulse pressure and the blood viscosity.

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1 ALFRED MARTINET: *"Pressions artérielles et viscosité sanguine."* Paris, Masson, 1912

**Organic hyposphyxia** has been observed by the writer:

1. In *acute or chronic tuberculous cases,* with the exception of those with added renal complications.

2. In *mitral disease, with or without compensation* Congenital or acquired mitral stenosis affords the most striking examples of this group. Doubtless the same applies to tricuspid stenosis

3 In the majority of cases of *chronic lung disturbance with emphysema and bronchitis.*

4. In *kyphotic patients*

5. In some cases of *uremia,* or more correctly, *azotemia*

**Functional hyposphyxia** has been very frequently witnessed. This type is almost the rule in young girls and a large number of women leading sedentary lives by choice or occupation (dressmakers, pianists, clerks, etc ), with low breathing capacities and weak musculatures The author has also come across it in a number of youths, scholars or students not interested in sports.

Often it is an inherited condition, dependent upon an actual, congenital and familial cardiovascular hypoplasia. In one such family, the grandfather, suffering from varicose veins, had always exhibited cyanosis and had cold, moist extremities; the mother, also varicose, was likewise hyposystolic and showed a high blood viscosity, the uncle had varicose veins and cyanosis of the face, lips, and extremities, an aunt, cyanotic and with varicose ulcers, was looked upon as having heart disease, another aunt was in a similar state, as for the patient himself, he was a cyanotic, sedentary individual with poor musculature, cold, moist extremities, and a congested liver, his pulse pressure was 30 millimeters and his viscosity 5 5.

This type of disturbed nutrition, accompanied by many other states of maldevelopment (dental, palatal, abdominal, etc ), frequently forms part of the symptom-group of *congenital syphilis.*

exists," he wrote, "the poorly nourished tissues and organs may actually undergo development, but remain small and weak The stunted individual that results does attain complete development, but without proper growth." (Henry Meige)

**From the standpoint of pathogenesis,** this syndrome constitutes, in last analysis, the outward manifestation of a circulatory disturbance characterized by *high venous pressure* with stasis dependent *either upon some obstruction in the left heart (mitral disorders)* or in the right heart (tricuspid disorders), or, as is more usually the case, upon a congenital underdevelopment of the heart (constitutional cardiac debility), or an obstruction in the lung (chronic tuberculous lung disorders), liver (cirrhosis or passive congestion), or veins (varicose veins, phlebitis, and cutaneous cyanosis)

Absolute or relative weakness of the cardiac contraction, peripheral circulatory weakness through vascular myasthenia, and respiratory weakness are met with in all these conditions

The above state of circulatory dynamism, with a low pulse pressure and high viscosity, points directly either to a congenital weakness or underdevelopment of the cardioarterial system (constitutional cardiac debility) or to an obstacle to the circulation behind the left heart (mitral valve, lungs, right heart, or liver). Venous plethora is the inevitable result and constitutes a mode of adaptation or defensive reaction to an unusual condition of the circulation

The presence of this syndrome in a chronic form, constituting an habitual circulatory state, **chronic hyposphyxia,** has been clinically *observed by the writer:*

1. In subjects with certain lesions, chiefly obvious cardiopulmonary conditions and corresponding with clearly defined nosologic entities, to which the term **secondary organic hyposphyxia** is applicable

2 In subjects apparently free of any organic heart or lung disorder so far described, the appellation **protopathic functional hyposphyxia** *(neurocirculatory asthenia)* may be used.

3. Occurrence of the syndrome as an acute or subacute, accidental and temporary condition, **acute temporary hyposphyxia,** has also been noted

# LOWER EXTREMITIES, PAIN IN.

The causal diagnosis of pain in the lower limbs is often obvious, as in rheumatic arthritis, post-infectious phlebitis, acute gouty attacks, etc.; yet sometimes it presents insuperable difficulties Few portions of the body are more accessible and readily examined; yet few are more complex, and none is the seat of pains that may be due to such a large variety of causes.

Any of the tissues of the extremity, bones, joints, muscles, veins, arteries, or nerves, may be the starting-point of painful affections; the spinal cord, vertebral column, various trophoneurotic disturbances, and various abdominopelvic disorders may likewise cause more or less obstinate pain in the lower extremities. Proper diagnosis sometimes demands an extremely painstaking clinical investigation and penetrating analysis

Any of the tissues, as we have seen, may be the starting-point of painful affections A succinct reference to each kind of tissue may prove serviceable:

I. **The Bones.**—*Traumatic conditions,* such as fractures, contusions, and sprains, generally self-evident, may be dismissed from the start, leaving for our consideration *osteoperiostitis,* osteomyelitis; osteosarcoma; an extremely common skeletal deformity, *flat foot,* which should always be kept in mind precisely because of its common occurrence, and the disorders of the bony spinal column, foremost among which is *Pott's disease.*

(*a*) **Osteoperiostitis**—This condition is characterized by the presence of a more or less localized painful area along the shaft of one of the bones of the lower limb, usually the femur or tibia, together with a variable degree of swelling. Osteoperiostitis may be

*Syphilitic,* as suggested by the history, recurrence of the pain at night (osteocopic pains), a positive Wassermann, and the efficacy of mixed treatment.

## LOW BLOOD-PRESSURE.

| | |
|---|---|
| **Anemias.** | Low cell count; pallor of mucous membranes; functional cardioarterial murmurs |
| **Hemorrhages.** | Traumatic or post-operative<br>(Progressive reduction of blood-pressure after trauma or an operation is always an indication of persistent hemorrhage). |
| **Neurasthenia.** | Neuropathic syndrome Headache, insomnia, constipation, asthenia, anxiety. |
| **Cachectic states.** | Cancer; senility, phthisic conditions |
| **Tuberculosis.** | Cough, fever, loss of weight, auscultatory signs<br>High blood-pressure should { either lead to exclusion of the diagnosis of tuberculosis, or lead to the detection of a renal complication |
| **Adrenal insufficiency.** | **Pathognomonic symptom-group:** Low blood-pressure, asthenia, and Sergent's white line<br>All grades { from Addison's disease or acute adrenalitis with rapid death, to the temporary and mild forms of post-infectious adrenal insufficiency |
| **Hyposphyxia.** | Slow circulation (low pressure, high viscosity), very often associated with pluriglandular insufficiency (hypocrinia).<br>(Pulse small and frequent, lividity, sensitiveness to cold, coldness and cyanosis of the extremities, venous plethora, etc ) |
| **Organic.** | 1 Tuberculous cases<br>2 Mitral cases<br>3 Chronic pneumopaths.<br>4 Kyphotics<br>5 Azotemics.<br>6 Congenital cardiovascular dystrophy (including congenital syphilis) |
| **Functional.** | Sedentary life, cardiomuscular debility, etc<br>Neurocirculatory asthenia. |

Whether the case be that of a child with unsteadiness of gait, weakness of the legs, more or less definite pains in the lower extremities, and impairment of general health;

Whether it be that of a subject whose parents have themselves detected a lateral deviation of the spinal column giving rise to pain;

Or whether, especially in an adult complaining of pain, an abscess pointing in the inguinal region brings definitely to light a Pott's disease which careful examination of the spinal column by inspection, percussion, motion, and fluoroscopy might have disclosed many months before.

(*f*) Lastly, one should bear in mind the *rare* possibility of an incipient *osteomalacia*, which would later be confirmed by characteristic deformities, with exaggeration of the normal curves of the bones, disordered locomotion, and pain on walking, becoming fatigued, or local pressure

II. **The Joints.**—It will not be necessary here to review all the possible causes of *joint pains*, a special section having already been included on this subject (see *Joint pains*). Systematic examination by inspection, palpation, mobilization, and if necessary fluoroscopy, will in the first place locate the pain in one of the joints of the extremity The special features of the joint disturbance, the history, onset, course, and simultaneous presence of other abnormal conditions will, as a rule, lead quickly to classification of the disorder in one of the following groups: Acute articular rheumatism, gonorrheal rheumatism, post-infectious rheumatism (scarlatinal, typhoid, etc.), or rheumatism due to some metabolic disorder (gout, arthritis deformans, etc ) Too much stress cannot be laid, here as elsewhere, upon the advisability of examining the seat of pain carefully and by direct inspection, of palpating and passively moving it, in short, of locating with care the pain and the seat of pathologic change, of precisely determining its nature, if possible, and of not resting content with the vague term "rheumatism," which is just as devoid of true diagnostic meaning as "headache" or "pain in the side"

Some joint involvements exhibit a rather pronounced selective tendency.

*Tuberculous,* though this form of osteoperiostitis is actually much less common than tuberculous osteoarthritis.

*Post-infectious, e g.,* post-typhoid; *staphylococcic* in the presence of recurring furunculosis or after sore throat.

(*b*) **Osteomyelitis.**—This is characterized by more severe and more diffuse pains and larger oscillations of temperature; it is generally post-infectious, *e g, typhoid* (post-typhoid) or *staphylococcic* (following sore throat or *furunculosis*).

(*c*) **Osteosarcoma.**—This is fortunately much less common, in fact exceptional, and is characterized by a rapidly progressive and generally painful enlargement involving the shaft of the femur. A mere mention of the condition would suffice were it not necessary to point out that it may sometimes be confounded with a *syphilitic gumma of the bone.* Indeed, in a case of sciatica resistant to all forms of treatment, in which the progressive development of a swelling of the femur had led to a diagnosis of osteosarcoma and a decision to amputate the limb, and the denials of the intelligent patient, answering questions in good faith, the absence of evidence of venereal disease, and the existence of nearly adolescent children free of any appreciable stigmata had seemed to warrant exclusion of the diagnosis of syphilis, the swelling was observed to melt away like butter before the sun's direct rays as a result of mercurial inunctions

(*d*) **Painful valgus flat foot** should be thought of in any adolescent complaining of pains in the legs and muscular contractures when he is fatigued, and the diagnosis may be made by the print method, which consists in having the patient place his feet over sheets of paper covered with lampblack. The footprints thus obtained show, in such cases, that the inner border of the foot is completely sagged down and that the foot is resting on the ground over the entire extent of the sole and not on its three normal pillars—posterior (os calcis), anterior (toes), and external (outer border of the foot).

(*e*) In this essential examination of the bony framework the spinal column, particularly in its dorsolumbar region, should not be neglected. This step in the examination is made necessary chiefly by the possibility of Pott's disease:

involved joints and painlessness. If the condition is only kept in mind, the diagnosis can be made by observation of the other indications of tabes—specific history, reflex disturbances (Argyll-Robertson pupil), loss of the patellar reflex, etc , astasia, abasia, ataxia, lightning pains, sphincter disturbances, etc

III. **The Muscles**—Disorders of the muscles, tendons, and serous membranes, occurring, respectively, in the form of myositis, tenositis, and bursitis, constitute possible localizations of rather indefinite painful processes the origin of which may be that described by Le Gendre, *viz.*, "a defective functioning of the locomotor apparatus either through lack of activity (sedentary mode of life) or through excessive activity (overstrain)," which renders it sensitive particularly to cosmic [meteorologic] influences that are normally not felt

Mention may here be made of the *myalgias,* often accompanied by *arthralgias* without objective manifestations, which, attended with an apparent typhoid state with sudden onset and albuminuria, frequently features the first or preicteric stage of primary infectious jaundice (hemorrhagic spirochetosis) Such pains may dominate the clinical picture sufficiently to mislead an inexperienced practitioner Thus, the author saw a fatal case of primary infectious jaundice which was admitted to a hospital on the third day of the illness with a diagnosis of "rheumatism" The patient, indeed, complained almost exclusively of pain and cramps in the thighs and a feeling in the knees as of constriction in a vice, without any redness or swelling but with a temperature of 40° C., a pulse rate of 136, albuminuria, a small liver, and incipient jaundice This case succumbed in ten days with the complete clinical picture of grave primary infectious jaundice—small liver, progressive jaundice, albuminuria, hemorrhages, and increasing hypothermia The pains in the muscles subsided as the jaundice grew more marked A few spirochetes were found in the blood and the urine

To complete the enumeration, mention may be made of the painful muscular spasms of nervous diseases, of tetanus, etc.

IV **The Veins**—Inflammations of the veins play a far more important rôle in pain in the lower extremities than they do in the

Gout very frequently occurs in the *joints of the great toe* (metatarsophalangeal joints).

Acute rheumatism of the *lower extremities* is located in the knees in four cases out of five. The same is true of gonorrheal arthritis.



Fig 809—Bilateral tabetic knee-joints. (*Glorieux* and *Van Gehuchten, Revue neurologique,* 1895).

Tuberculosis involves almost indifferently any of the joints; yet its predilection for the knee (white swelling) and the hip (coxalgia) is well known.

*Malum coxæ senilis,* the pathogenesis of which is as yet obscure, involves, as the term implies, the hip in *elderly subjects.*

Nor should one forget the characteristic *tabetic* or *Charcot joints,* with the attendant marked deformity, extreme laxity of the

induration around the vein, and the presence of trophic changes of the skin lead to the proper diagnosis in chronic forms. In acute and subacute phlebitis, the history (as of infection, a surgical procedure, or parturition), the fever, edema, special sensitiveness

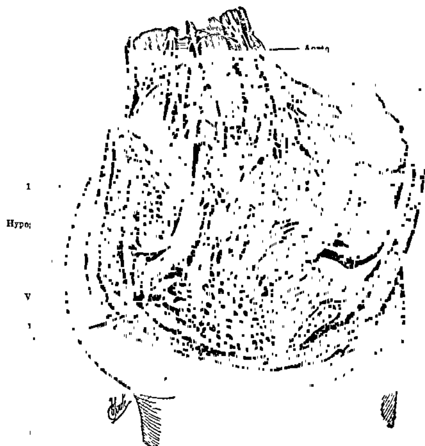


Fig 811—Lumbosacral and hypogastric plexuses in the female (sources of the sciatic nerve) (*Hirschfeld*)

along the course of the vein, and sometimes the finding of a hard, cord-like vein insure recognition of the existing disorder

V. **The Arteries.**—Pain attending disease of the arteries in the lower extremities is not of very frequent occurrence, but when present is very obstinate It is the result of arteritis of the femoral or of the tibial or peroneal vessels Arteriosclerosis,

case of pain of the upper limbs. Here *phlebitis* is much more common, being either of *chronic* nature, as in *chronic degenerative phlebitis* (*varicose veins*) or *acute or subacute infectious phlebitis*,



Fig 810—Lumbosacral and hypogastric plexuses in the male (sources of the sciatic nerve) (*Hirschfeld*).

the latter especially post-infectious, post-operative or puerperal in origin. The diagnosis of the condition is, as a rule, easy. The mere finding of dilated veins, which in the chronic forms are particularly prominent with the patient in the standing posture, the observation upon palpation of phlebosclerosis and frequently of

(posterior tibial nerve) and extends to the plantar surface of the foot, nearly the whole of which is innervated by it.

Brief reference to these anatomic facts was necessary because:

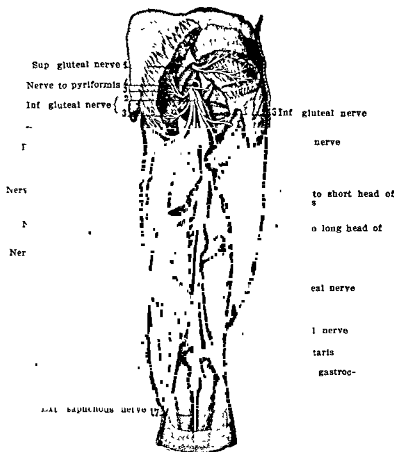



Fig 812—The greater sciatic nerve (*Sappey*) The small sciatic nerve consists of the two small trunks designated above as the inferior gluteal nerve and the posterior cutaneous nerve of the thigh

1 The sciatic nerve must be investigated in every portion of its course, from the lumbosacral vertebræ to the os calcis and the sole of the foot. There are certain points which, on account of their regional anatomic relationships, lend themselves especially well to examination and the eliciting of tenderness. Valleix made a special study of these elective points, whence the designation

gout, specific disease, and infections, especially typhoid fever, are by far the most frequent causes—in fact, the only ones met with in the author's experience.

As serviceable diagnostic indications of such a condition the following features may be mentioned:

A distinct difference of blood-pressure in the two limbs; gradual diminution of the beats below the involved portion of the artery, intermittent claudication (in the chronic forms); lowered local temperature of the affected limb; coldness and pallor; sometimes, where the vascular distribution from a certain trunk is actually obliterated and no collateral circulation forms, hypothermia, vasomotor disturbances, and cyanosis occur and are followed by the appearance of areas of necrosis of varying extent, which may later result in mutilation of the part.

VI **The Nerves.**—The sciatic nerve is the one by far the most frequently involved in the lower extremities One should remember that the course of the sciatic nerve comprises a *spinal portion*, originating in the anterior branches of the last two lumbar and first four sacral nerves; a *pelvic portion*, in which the sacral plexus, formed by the convergence and subsequent fusion of the foregoing nerve-roots, enters into direct or indirect anatomical relationship with nearly all the pelvic organs; a *gluteal portion*, beginning at the great sacro-sciatic notch, from which the single, combined trunk of the sciatic issues at the gluteal fold, being ensconced in this region in a musculo-osseous recess bounded within by the ischium and externally by the greater trochanter; a deep, intramuscular *femoral portion*, in which the nerve is lodged in a vertical muscular trough bounded externally by the long head of the triceps and internally by the semitendinosus and semimembranosus, and finally, a *terminal portion*, in which it divides, four fingerbreadths above the plane of the tibiofemoral joint, into its two terminal branches, the *external popliteal* nerve, which, after having passed around the external condyle of the femur and the inner surface of the head of the fibula, continues through the thickness of the peroneus longus and along the external and dorsal aspects of the foot, and the *internal popliteal* nerve, which crosses the popliteal space obliquely, passes above the soleus

examined just as carefully to discover the cause as from the symptomatic standpoint.

Along the spinal portion of the sciatic *bony changes* should especially be looked for, *viz*, Pott's disease, exostoses, spondylitis, gumma, or cancer of the vertebræ; and likewise *meningeal disturbances*, such as acute or chronic meningomyelitis or the common condition termed meningeal hyperemia. An undemonstrable, but seemingly probable and frequent cause, judging from the percentage of cases in which the author found it and the therapeutic efficacy

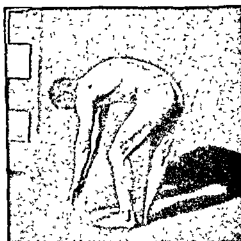


Fig 814—Left-sided sciatica. Forward bending of the body is possible only if the knee of the affected side is flexed (*G Roussy*)

of hamamelis in large doses, is intraspinal venous hyperemia or the "intraspinal varicose state," causing pressure upon and strangulation of the nerve-roots upon their emergence from the bone (see *Lumbar pain*).

Along the pelvic portion of the sciatic, disorders of the rectum, prostate, bladder, Fallopian tubes and ovaries, and uterus may be and frequently are the source of sciatic neuralgia or neuritis through one of the four following factors. 1. Pressure, as by primary new growths or secondary glandular enlargements. 2. Hyperemia, as in inflammatory pelvic congestion or acute congestion of hemorrhoids 3 Reflex irritation from a remote structure, as in urethritis or orchitis 4 Direct involvement in malignancy or inflammation, as in tumor of the rectum or uterus.

*"Valleix's points"* which has remained attached to them. These points are shown in the annexed diagram.

Mention should be made of *Lasègue's well-known, simple and rapid maneuver,* which consists in flexing the thigh on the pelvis with the leg extended During this procedure the sciatic nerve, after running a practically straight course with the limb in complete extension, is forced into a sharp curve in its gluteal course by the flexion of the limb, and is thereby stretched like a violin string when the bridge is raised into position. The resulting tension at once excites a characteristic pain along the nerve in the presence of sciatica.

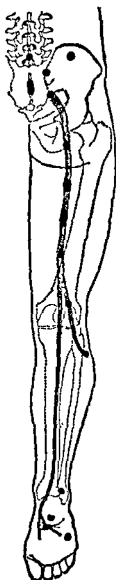


Fig 813—Valleix's points on the posterior aspect of the lower extremity, and their relationship to the bony skeleton.

Lasègue's test is merely the simplest and most commonly used of the various procedures of *stretching the sciatic,* which brings on pain where there is disorder of the nerve. It is plain that any form of motion, whether active or passive, which causes stretching will excite the same sort of pain or a characteristic posture of the limb having for its purpose to obviate the stretching of the nerve. When the patient is standing and is asked to pick up an object from the floor, keeping the legs extended on the thighs, he will instinctively and necessarily flex the involved limb or move it backward in order to avoid tension on the nerve

When the patient is recumbent and is requested to sit up, keeping his legs straight, he will similarly, and for the same reason, flex the affected limb (*Sicard's "raised knee sign"*), thus exhibiting what appears as a unilateral Kernig sign (G. Roussy).

2 The origin of the nerve pain may be at any point along the course of the nerve. The whole course of the nerve should be

temperature, absence of local inflammation and of signs of tabes dorsalis, and sometimes the steppage gait in an inveterate alcoholic.

If still nothing can be found, the condition may be labelled a simple, primary, or idiopathic sciatica, the physician thus escaping the necessity of committing himself concerning the actual nature of the disturbance.

It is plain that, apart from symptomatic, palliative treatment of the neuralgia, which, indeed, is frequently all that is required, an accurate causal diagnosis can alone supply a reliable basis for curative treatment.



Fig. 816—Right-sided sciatica. The patient, when in the sitting position, is unable to extend the affected limb completely (*G Roussy*).

One should always carefully examine for muscular atrophy and electric reactions, which afford a distinction between neuralgic sciatica, ordinarily a mild condition, and neuritic sciatica, which is always serious and sometimes incurable

* * *

The lumbar plexus, formed by anastomoses of the anterior divisions of the last four lumbar nerves, and its branches—the ilio-hypogastric, ilio-inguinal, external cutaneous, genito-crural, obturator, and especially, the anterior crural—is, apparently, much less frequently affected than the sciatic. Yet *lumboabdominal neur-*

From the above considerations the importance of a systematic examination of the pelvic structures in the presence of sciatica will readily be seen.

**Along the femoral portion** *of the nerve, in the buttock,* the conditions oftenest met with as exciting causes of sciatica are *traumatism* (falls, sudden impacts or blows) and *coxofemoral arthritis and periarthritis.* In the femoral region a special search should be made for factors resulting in compression, chiefly by bone, such as osteoperiostitis, gumma, and osteosarcoma; in the *popliteal space,* the



Fig 815—Right-sided sciatica. In some instances, when the body is being bent forward, the patient spontaneously tilts the affected lower limb backward (*G Roussy*).

commonest exciting causes are crypts, aneurysm, and fungous joint inflammation.

Where, however, all these local causes of neuralgia and neuritis can be excluded, an inquiry should be made for general causes, some of which are still rather obscure:

Neuralgia *a frigore* (following exposure to cold).

Rheumatic (?) neuralgia.

Diathetic neuralgia; diabetes should be remembered as a frequent cause of obstinate sciatica.

*Post-infectious neuralgia*

Toxic neuralgia; in this connection special attention should be paid to *alcoholism,* with the attendant diffuse pains, lack of febrile

tions in the pelvis, especially those involving the Fallopian tubes or the ovaries.

Lastly, mention should be made of the *lightning pains* of tabes dorsalis, exhibiting definite features in their paroxysmal occurrence, lancinating, fulgurant, and boring character, but the diagnosis of which should, in short, be based mainly on the major symptom-group of tabes, *viz.*, specific history; reflex disturbances, such as

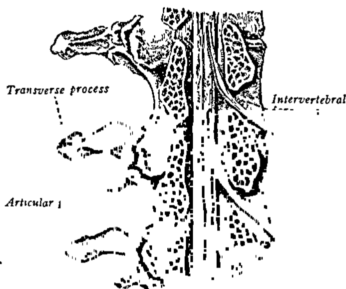



Fig 818—Relations of the intervertebral foramina of the lower lumbar region with the lumbar spinal ganglia

loss of the patellar reflex and the Argyll-Robertson pupil; disturbances of station and equilibrium (astasia), disturbances of motion (ataxia), sphincter disturbances, etc.

* * *

In a striking synthetic study concerning cases of *neurodocitis* and *vertebral funiculitis*, Sicard (*Presse méd.*, Jan. 7, 1918), gave a good general account and excellent anatomic classification of the changes occurring in the spinal nerves from their point of issue in the spinal cord to their rearrangement in separate fascicular groups. A good diagram condensing the essential facts in this connection seems adequate at this point. Neurodocitis is a general term desig-

*algia* with its painful points (lumbar, iliac, abdominal and scrotal), frequently encountered in pyelonephritic disorders; *external cutaneous neuralgia* with its superior interiliac painful point, and especially *anterior crural neuralgia,* with its painful points dispersed along the anterointernal aspect of the thigh, leg, and foot (tender points in

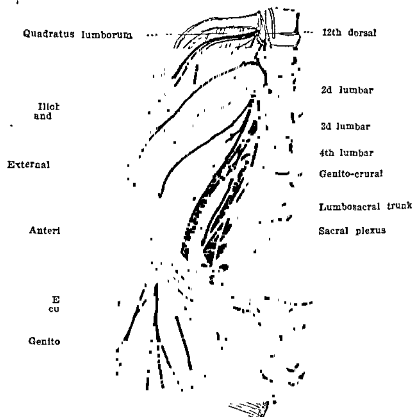



Fig 817.—The lumbar plexus

the inguinal region, the crural region, over the internal condyle, the internal malleolus and the inner margin of the foot) are not very uncommon, particularly that last mentioned

Lumboabdominal neuralgia, as we have just seen, is an almost constant clinical appurtenance of many pyelonephritic infections, especially renal colic

In anterior crural neuralgia a special examination should be made for Pott's disease, psoas inflammation, appendicitis, typhlitis, inguinal and femoral hernia, and in a general way for affec-

However long and tedious the preceding enumeration may have been, it constitutes only an incomplete list of the local disorders; yet it includes the majority, at least, of the painful conditions met with in the lower extremities. (In addition there are to be thought of such discomforts as may arise from tight leggings, corns, perforating ulcers, ill-fitting footwear, abscesses, lymphangitis, suppurative processes and the corresponding glandular enlargements, etc.—all self-evident local disorders.)

As a parting piece of counsel, it may be stated that whatever variety of pain in the lower extremity is complained of, one should never fail to examine:

(*a*) The spinal column.
(*b*) The hip joint.
(*c*) The region of the appendix.
(*d*) The kidney region.
(*e*) The urine

nating the changes resulting from compression of certain nerve-trunks in the natural osseous, fibro-osseous, or fascial canals. The word funiculitis is here taken to signify a neurodocitis of the tissues surrounding the intervertebral foramina

Ordinary sciatic neuralgia is either a neurodocitis resulting from compression of the nerve at the great sacrosciatic notch, in

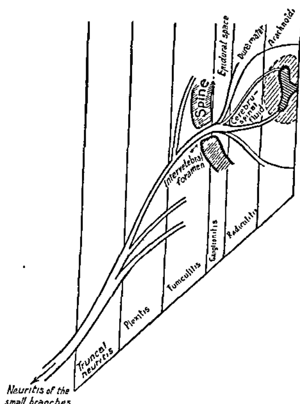



Fig 819.—The several sections of the nerve paths from the spinal cord to the periphery Ordinary sciatica is a funiculitis at the intervertebral foramen

the ischiotrochanteric space, or on the outer aspect of the fibula, or a funiculitis of the intervertebral foramen of rheumatic, gouty, or arthritic origin

True lumbago is a bilateral rheumatic funiculitis involving the 2d, 3d, and 4th lumbar nerves.

*Syphilis* usually causes radiculitis and myelitis, while tuberculosis and cancer oftenest lead to funiculitis.

versely, an obvious case of Pott's disease of the lower dorsal region with abscess formation labelled lumbago.

Such mistakes may be accounted for in many ways

First and foremost is the unfortunate habit of not examining the painful region directly and by inspection Only exceptionally, indeed, is the lumbar region actually subjected to a systematic examination in a patient complaining of "kidney pains."

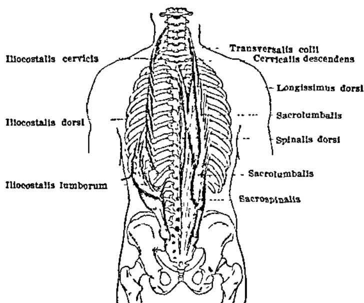



Fig 821—The spinal muscles

As a matter of fact, there are few regions of the body that demand a more thorough and systematic local examination, since very few regions exhibit painful manifestations originating in such diverse and variously situated disturbances. While one of the anatomic peculiarities of the region is the presence of the thick sacrolumbar masses of muscular tissue, which act, to all intents and purposes, as the muscles governing the erect posture and are so frequently rendered painful by the most varied pathologic states, the following regional anatomic divisions should be kept in mind

1. Muscular region: The sacrolumbar mass of muscle tissue.
2 The bony spine and the sacroiliac joints.

# LUMBAR PAIN. BACKACHE.

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Few symptoms are so frequently misinterpreted as lumbar pain, backache, or, as popularly termed, "kidney pains" The

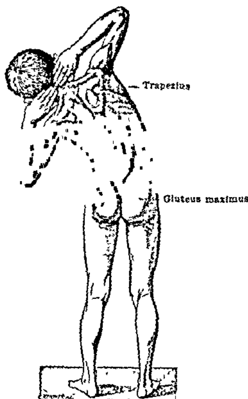



Fig 820—Lumbar musculature The spinal muscles on the side opposite to that of flexion are in contraction The transverse folds of skin over the spinal muscles on the relaxed side are readily seen (*P. Desfosses*).

author has personally seen—an instance almost unbelievable, yet absolutely authentic—a case of ordinary lumbago labelled Bright's disease (needless to state, examination showed neither albumin, casts, high pressure, *nor any increase in blood urea*), and con-

tion that might without impropriety be placed in the group of "rheumatic disturbances the result of defective functioning of the locomotor apparatus," masterfully described by Le Gendre.[1]

Undoubtedly there occurs, moreover, an *acute* or *subacute lumbago of rheumatic origin* which is very favorably influenced by sodium salicylate.

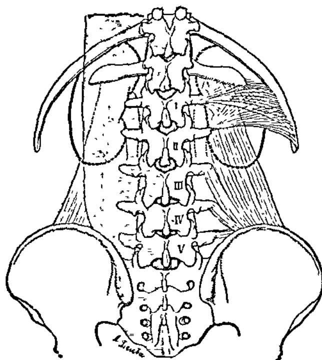



Fig 823—Posterior relations of the kidneys On the left side is seen the main mass of spinal muscle (shaded), and projecting beyond it below, the quadratus lumborum On the right side the main spinal muscle mass has been removed, exposing the quadratus lumborum and the lumbocostal ligament

(*c*) Subacute or chronic lumbago of *psychoneurotic cases*, apparently the local clinical expression of an actual constitutional neuromuscular asthenia, and which may occur either as: 1. A localized pain constituting an actual topoalgia 2 A regional dysesthesia, with morbid sensations of pressure, of sharp, fleeting pains, of heat, or of cold, frequently with abnormal sensitiveness of the region to pal-

[1] LE GENDRE: *Acad de méd*, May 9, 1911.

3. The spinal cord and nervous system.
4. Paravertebral organs: The aorta and lymphatic structures
5 The abdominal viscera, including especially the kidneys, spleen, liver, colon, and uterus.

When a patient comes complaining of "kidney pains" or backache, the lumbar region should be exposed and a systematic examination of the structures above enumerated proceeded with

1. The skin.—This is sometimes the seat of herpes zoster (zona) covering a varying extent of surface.

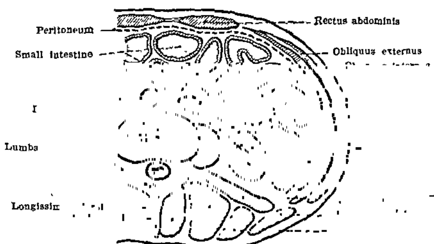



Fig 822—Transverse section through the lumbar region

2. The sacrolumbar muscular system.—Palpation and inspection with the patient in various positions, especially the erect posture, together with movements of anteflexion, extension, torsion, and lateral bending will often lead to the discovery that the seat of pain is actually in the muscle tissue, the condition being a true lumbar muscular pain for which, seemingly, the term lumbago should be set apart, but which may yet be encountered under very variable clinical circumstances, to wit·

(a) Acute lumbago, following a forceful straightening of the flexed body, as in lifting a heavy weight, such as a trunk.

(b) Subacute lumbago, following a prolonged march, with fatigue and exhaustion. This represents a "forcing" of the muscle, which becomes painful because of overwork,—a condi-

fever, etc. The condition is, in short, a *vertebral osteoarthritis*, or a *deforming* or *post-infectious lumbarthritis*. Nor is post-traumatic spondylitis a rare affection.

The limitation of motion, the stiffness in the lumbar region, the pain induced in the lumbar spine by flexion or torsion of the body, the cracking sensations experienced by the patients themselves, the chronic or subacute course of the disorder, the history, and especially the x-ray examination, will lead to the diagnosis.[1] The disorder particularly to be excluded in these cases is *Pott's disease.*

(*b*) **Pott's disease**, the recognized evidences of which need here scarcely be recalled Localized pain, most marked in one vertebra; radiation of pain to the lower extremities; cessation of pain upon rest in recumbency and immobilization of the affected region; and ultimately, angular deformity at the point of involvement and paretic disturbances of the sphincters and lower extremities, with exaggerated reflexes, abscess formation, etc.

*Pott's disease should always be thought of in the presence of chronic lumbar pain.*

(*c*) **Iliac and sacroiliac osteoarthritis**, the location of which is determined by careful palpation.

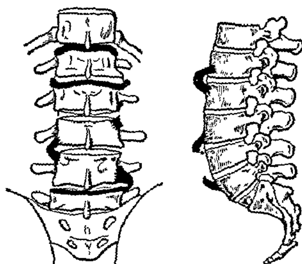
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[1] Béclère, quoted by Léri (*Presse méd*, Feb 28, 1918), had already published in 1906 the following differential table relating to chronic rheumatism of the vertebræ and rhizomelic spondylosis

| 1. Chronic vertebral rheumatism | 2 Rhizomelic spondylosis |
|---|---|
| First feature. Distortion of the bodies of the vertebræ consisting of a broadening of the upper and lower surfaces and exaggeration of the circular concavity | First feature No distortion of the bodies of the vertebræ, which are almost cylindrical in shape |
| Second feature The intervertebral discs are distinctly more transparent than the bodies | Second feature The discs are not more transparent than the bodies |
| Third feature Little or no suggestion of a vertical opaque band corresponding to the ligaments is present. | Third feature Both the bodies and discs are covered by a broad band with parallel borders; the outermost portions of the vertebral bodies project beyond this band; the processes of the vertebræ exhibit a remarkable and unusual degree of transparency |

pation, the whole constituting a vague neuromuscular syndrome. 3. Deep pain, weakness, and exhaustion with lumbar muscle pains following strong emotional impressions; an actual paroxysmal emotional lumbago superimposed upon a chronic psychoneurotic lumbago attending habitual asthenia.

The absence of definite local lesions, the history, the neurotic stigmata, the habitual psychasthenia, the chronic course of the morbid manifestations, and their recrudescence after emotional



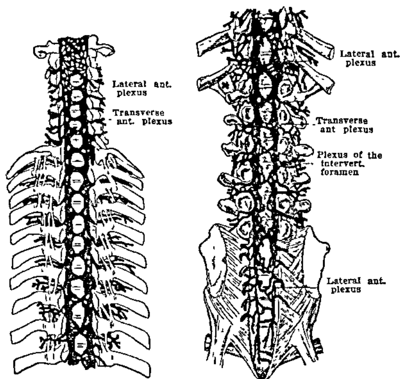
Figs 824 and 825—Osteospondylitis of the vertebræ

impressions are the most reliable diagnostic features of these cases.

3 The **spinal column**, and more particularly the *lumbar vertebræ*, with which we are here especially concerned, may be the seat of many pathologic conditions causing lumbar pain

(*a*) First and foremost should be placed the chronic inflammatory states or **spondylitis** of the vertebræ, their periosteum, and the intervertebral joints, leading to the exostoses, adhesions, and ankyloses *frequently encountered in sedentary individuals after the fourth decade of life* Some rough sketches illustrating these conditions are presented herewith Such instances of *spondylitis* are met with among all *the usual cases of arthritis deformans*, and post-infectious rheumatism, *e g.*, after *pneumonia*, *tonsillitis*, *typhoid*

showing daily, almost periodic exacerbations; these cases were, as a matter of fact, greatly relieved by counterirritation with fly blisters and wet cupping over the lumbar region and medication directed toward overcoming the venous congestion, *viz.*, adrenalin, strychnine, and hamamelis. This condition must play an important rôle in the lumbago of psychoneurotic subjects.




Figs 826 and 827 —Intraspinal venous plexuses

(*f*) Lastly, it should not be forgotten that the spontaneous or artificially induced pain in *some cases of sciaticalgia* may ascend above the great sacrosciatic notch to the vicinity of the sacroiliac joint or of the transverse processes of the lumbar vertebræ and the lumbosacral masses of muscle tissue

Examination for Lasègue's sign is of the greatest service in these cases

In the frequent instances of sciatica combined with kyphoscoliosis or scoliosis, hyperesthesia and hyperkinesia of the lum-

4. **Inflammatory conditions of the spinal cord and vertebral column** cause well-known forms of *backache*. They are met with chiefly in the following disorders.

(*a*) In **acute meningomyelitis** (acute meningitis; cerebrospinal meningitis), the diagnosis of *which is based on the simultaneous presence of constitutional signs of infection, such as fever and leucocytosis, in conjunction with the meningeal symptom-group* (headache, backache, Kernig's sign, etc.), and is confirmed by examination of the cerebrospinal fluid, showing an excess of lymphocytes or polynuclears, the presence of meningococci, etc.

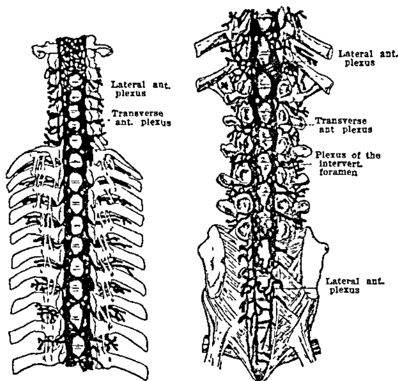
(*b*) The **onset of many infectious diseases,** particularly influenza, smallpox, and pneumonia, is, as is well known, frequently marked by extremely severe backache. Examination of a number of specimens of cerebrospinal fluid obtained under these conditions led to the surmise that the beginning of these disorders is often accompanied by a sharp but temporary meningeal congestion, which later passes off along with the backache which is its clinical manifestation.

(*c*) Mention may here be made of an expression used in military medicine, *viz.*, *backache with fever* (*courbature fébrile*)—on the whole a rather happy expression since it combines both the symptoms, *backache* and *fever*, which feature the clinical state to which it refers. Unquestionably this term comprises a number of different conditions and careful investigation would lead frequently to the discovery of cases of "incomplete" paratyphoid or even typhoid cases On the other hand, it is certainly true that if the clinical entities now recognized are eliminated from it there remains a large percentage of undetermined, cryptogenic infections, usually transient and mild, but for which an accurate designation would be hard to find

(*d*) The same sort of a *sharp, temporary* **meningeal reaction with backache and fever** has been noted by the author in **certain cases of secondary syphilis** running an acute, febrile course

(*e*) The author has been led to consider as symptomatic of *venous congestion of the spinal and perispinal plexuses* certain instances of **chronic lumbago** in subjects suffering from hemorrhoids, with high venous pressure and low arterial pressure, the lumbar pain

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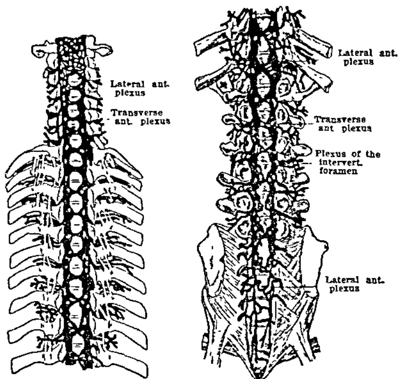
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ward or along the iliohypogastric and ilioinguinal nerves, extending around the body.

"In other instances, it radiates even to the neck of the bladder or the spermatic cord, as in stone, a fact accounted for by the existence of the genital fibers of the ilioinguinal nerve.

"The pain may be located *sympathetically* (Guyon's reno-renal reflex) in the opposite kidney, thus bringing in a disturbing factor in the interpretation of the case. A patient with a stone in the right kidney often experiences pain in the left kidney. The same sort of thing is observed in renal congestion.

"The pain may be either slight, dull, and deep-seated or occur in paroxysms introducing the symptom-group of renal colic (hydronephrosis) or of nephritic colic (descent of a stone), *while generally wanting in nephritis, in cancer, and in some common forms of renal tuberculosis, it is present especially in certain cases of movable kidney, in hydronephrosis, and in lithiasis, in which it is increased by walking, riding in vehicles, violent exercise, and motion in general.*"

**Physical examination** of the kidneys, which should be combined with the study of the functional signs, consists chiefly of *palpation* and the finding of either a movable, a distended, a subcostal, or a cancerous kidney, the organ, in the latter instance, being often hard, irregular, and mobile

Such a clinical investigation for tumor in the hypochondriac regions is sometimes hard to interpret, on account of the presence of the liver on the right side and the spleen on the left

By this procedure, however, one is enabled to ascertain the exact position of the enlarged kidney, whether thoracic or subcostal, its mobility, its smooth or lobulated surface, its consistency (hard or soft; fluctuation in hydronephrosis), and its irregular shape (in certain instances of malignancy).

Among the further sources of information that may be availed of by palpation are the three *ureteral points of tenderness, viz.,* the superior or *paraumbilical* point, corresponding to the renal pelvis; the intermediate or *iliac* point, corresponding to the crossing of the external iliac vessels, and the inferior or *vesical* point, corresponding to the interstitial, intravesical portion of the ureter, palpable

bosacral muscular mass are constantly present. It is noteworthy, however, that this hyperesthesia and hyperkinesia are in some instances homologous (*i.e*, present on the same side as the sciatic pain), while in others they are on the opposite side; no reliable explanation of this fact can at present be vouchsafed.

This peculiarity is of importance in the exposure of malingerers. Where, in conjunction with the symptoms of sciatica, there is noted spontaneous or induced hyperesthesia of the lumbar region on the opposite side, one may almost certainly, unless

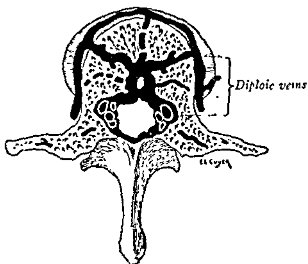



Fig. 828—Diploic veins in the body of a vertebra

the subject is thoroughly familiar with the clinical features of sciatica, exclude the possibility of malingering.

5. The popular expression "pain in the kidneys" correctly suggests the anatomic relationships of the kidneys to the lumbar regions. It is an actual fact that many renal disorders are accompanied by lumbar pain, the following succinct description of which is borrowed from Cathelin:

"The first indication of reno-ureteral affections which plays a predominant rôle in the patient's own estimation is *pain*, which may be either spontaneous or artificially brought on by motion or by pressure in the costovertebral angle, particularly at the apex of this angle. It is seldom present anteriorly, yet radiates either in the direction of the ureter obliquely downward and in-

ward or along the iliohypogastric and ilioinguinal nerves, extending around the body.

"In other instances, it radiates even to the neck of the bladder or the spermatic cord, as in stone, a fact accounted for by the existence of the genital fibers of the ilioinguinal nerve.

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through the vagina in women and through the rectum in the male subject.

6. **Abdominal ptosis,** *various uterine disorders,* and *pregnancy* are the cause—frequently obvious—of many instances of lumbago The mere thought of them is often sufficient to make plain their causal relationship to the lumbar discomfort. The author has seen very many cases of obstinate lumbago yield to the wearing of a suitable abdominal belt.

7. **Exceptional forms.**—Mention may be made of certain truly exceptional causes of lumbar pain, *viz*, **cholelithiasis, aortic aneurysm, and vertebral and paravertebral tumors.** The presence of a painful point in the right lower lumbar region with contraction of the muscles of the posterior wall in attacks of acute appendicitis has been pointed out by a number of authors.

* * *

Brief clinical examination will often discover very easily the actual cause of lumbar pain. The following few questions are sometimes conclusive in this connection:

1 **How long has the patient been suffering from backache?**

(*a*) The patient may have had it for a few days, the onset having been sudden, after exertion or a forced march. *True or rheumatic muscular lumbago*

(*b*) He may suffer from it in chronic fashion, with exacerbations, and exhibit an asthenic appearance· *Depressive psychoneuroses, spondylitis, Pott's disease, spinal and perispinal venous congestion, chronic abdominal affections, or chronic nephritis.*

2. **The backache is closely accompanied by an infectious disorder:** *Meningomyelitis, onset of infectious diseases* (such as influenza, small-pox, pneumonia, etc ), *backache with fever,* etc

3. **The lumbar pain is increased by the standing posture and by flexor and extensor movements of the body:** The seat of pain may be in the spinal column (*Pott's disease, vertebral osteoarthritis,* etc.) or the lumbosacral muscle mass (*traumatism, strained back, rheumatism*)

4. **Is there pain on pressure or percussion, and if so, where?** In a vertebra: *Vertebral osteoarthritis, Pott's disease.*

In the costovertebral angle: *A renal disorder.*

In a sacroiliac joint: *Sacroiliac osteoarthritis.*

Involving the last lumbar nerves and along the sciatic nerve: *Sciatic algia*

5. Does the urine contain albumin, blood, or pus? *A renal disorder.*

# NECK, SWELLINGS IN THE.

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The limits of that which constitutes the neck are rather hard to define. **Anteriorly,** it is bounded below by the angle formed by the clavicles and sternum, above by the lower jaw and an imaginary line extending from the angle of the jaw to the mastoid process. **Posteriorly,** it is bounded above by the mastoid processes and the external occipital protuberance, while below its limits are indefinite; it forms a continuous surface, in the absence of any prominent landmarks, with the dorsal region This constitutes the nuchal region, which is one of subsidiary importance from the medical aspect The anterior and lateral surfaces of the neck, on the other hand, are frequently the seat of swellings concerning which the practitioner's opinion is often in demand, and although by far the greater number of these belong rather to the domain of surgery, it seems essential to refer to them briefly The subject being, however, somewhat out of the exact limits of the field covered by this work, the data presented will be given merely in a brief, suggestive form, with just sufficient detail properly to recall the various possibilities to the reader's mind

For convenience of description, it is advisable to divide the anterolateral region of the neck into two median parts, the supra- and infra-hyoid, and two lateral parts, the sternomastoid and the supraclavicular, as represented in Fig. 829.

* * *

**The patient comes to seek the practitioner's advice:**

*Either on account of a local or diffuse swelling in the neck, of an acute and painful* **inflammatory type.**

*Or on account of a chronic enlargement of* **non-inflammatory nature.** A swelling in the neck may even be discovered by the physician in the course of a systematic examination without the patient having previously been aware of its presence.

The acute inflammatory swellings in the neck comprise adenitis, thyroiditis, and cellulitis of the neck.

Submaxillary adenitis, a frequent condition in children, is generally of dental origin

Adenitis below the angle of the jaw either originates from the pharynx or occurs in conjunction with disturbances localized in the *wisdom tooth.*

In suprahyoid adenitis a primary inflammatory condition should be examined for in the lower lip and suprahyoid superficial




Fig 829—Anterior surface of the neck, showing the regional subdivisions and underlying structures

tissues. Allied to this is the serious condition known as *suprahyoid cellulitis,* following Ludwig's angina, the primary source of which is an inflammation of the floor of the mouth in the form of a sublingual abscess.

In the infrahyoid region, an inflammatory swelling is necessarily associated with laryngeal manifestations such as aphonia, dyspnea, and suffocative attacks It follows an abscess of the thyrohyoid and thyroepiglottic spaces, detected by palpation from the pharynx: On following down the base of the tongue, an edematous, tender, and sometimes fluctuating swelling is found in front of and on the lateral surfaces of the epiglottis

**Sternomastoid adenitis** is almost inevitably accompanied by *symptomatic torticollis.* The source of infection is to be looked for in the regions of the *mastoid* (mastoiditis) and of the *pharynx* (phlegmonous angina).

**Diffuse cellulitis of the neck** *sometimes follows the form of* adenitis referred to; much oftener, however, it develops directly, in a predisposed subject, as a result of some infection of the mouth or pharynx.

**All of the above conditions are mainly surgical.**

**Thyroiditis** is chiefly a medical disorder. It appears in the form of a uni- or bi- lateral swelling in the infrahyoid region representing the thyroid gland, the shape of which is reproduced thereby, the enlargement is sensitive rather than painful, of elastic consistency, and covered with healthy skin of normal hue but crossed by dilated veins. The condition is accompanied by dyspnea, cough, a rough quality of the voice, and sometimes by frothy, blood-stained expectoration The pain is worse on swallowing The disorder develops either spontaneously, with a sudden onset marked by a chill and fever, or in consequence of an infectious disease. Resolution is the rule, and suppuration exceptional.

* * *

**Chronic Enlargements in the Neck.**—These are by far the *more important from the medical standpoint.*

A regional classification is necessary for practical clinical purposes · A swelling may exist either on one of the lateral aspects *of the neck or in the median region*

**Enlargements on the Lateral Aspects of the Neck.**

1. **Glandular enlargements**.—Here again, involvement of glands supplies the greatest number of enlargements.

The usual causes of such enlargements are **tuberculosis, syphilis, lymphatic hypertrophy,** and **neoplasm**; the main diagnostic features of these conditions have already been given under *Glandular enlargements (q.v.)*

**Softened tuberculous lymph-nodes** are identified by their fluctuation and non-reducibility. Where there is periadenitis,




Fig 830—Lymphatics of the head and neck (*Sappey*).

*1, 1* Lymphatic vessels leading to the parotid nodes *2, 2* Inferior frontal lymphatics *3, 3* Superior frontal lymphatics *4, 4* Parietal lymphatic vessels, these pass vertically downward, anastomosing with the neighboring vessels, and end in the mastoid nodes *5, 5* Origin of these vessels *6, 6* Anterior suboccipital vessels, converging to form a single trunk which, after a prolonged course, terminates in one of the lowest cervical nodes *7* Trunk resulting from coalescence of these vessels *8* Node in which this trunk terminates *9, 9* Posterior suboccipital vessels, terminating in two nodes located at the anterior border of the trapezius muscle *10, 10* The [illegible] nodes *11* Large horizontal trunk start-
ing
mus
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ing
*17.*
of the lower lip *19* Suprahyoid node in which the above vessels terminate *20.* Large lymphatic vein

with adhesion to the reddened and tense overlying skin, the diagnosis is extremely easy.

The *differential diagnosis between tuberculous lymphadenitis and lymphadenoma* sometimes occasions difficulty: "These two forms of enlargement at first present common features. They may be of the same size and shape, in either case consisting of several lymph-glands, they are movable in respect of the skin and

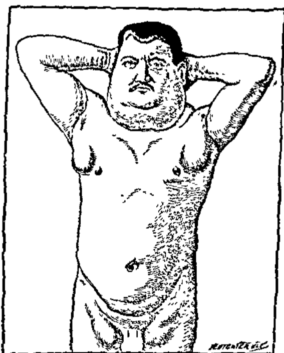


Fig 831—Adenolipomatosis (*Launois* and *Bensaude*).

deeper tissues, though in some instances fixed by contraction of the sternomastoid muscle; they have the same seat of predilection (angle of the jaw, submaxillary region, and carotid chain); they may be symmetrically placed, and also appear at the same age, though scrofulous adenitis is more frequent in early life. Yet each of the conditions has its own particular features. Thus, lymphadenoma is relatively uncommon, while tuberculous adenitis is by far the commonest form of enlargement met with in the neck. Lymphadenoma is of an even consistency and imparts to the fingers a sensation similar to that of renal parenchyma.

Tuberculous adenitis, on the other hand, may be of uneven consistency and present some hard spots and other areas of softening. Tuberculous adenitis undergoes suppuration, while lymphadenoma never does. Consequently, whenever fluctuation, or even periadenitis, is detected, or a sinus or the scar of a former

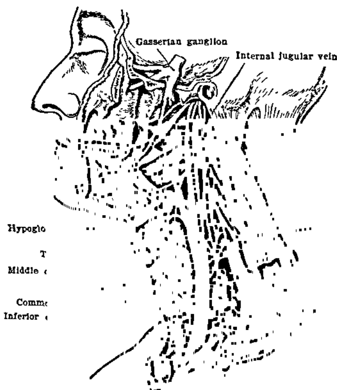



Fig 832—Deep structures of the neck (vessels)

abscess observed, a definite diagnosis of tuberculous adenitis may be made. Finally, although lymphadenoma affords only negative signs, since at one period the same evidences are found as in lymphatic tuberculosis, it should be borne in mind that lymphadenoma may be diagnosed where an enlargement of considerable size is present; where lymph-nodes similarly affected are found in the axillæ and the inguinal and other lymphatic regions; where an enlargement of the same nature is found in another organ, either the testicle or the skin (mycosis fung-

oides); where enlargement of the spleen, tonsils, or thyroid gland is simultaneously found, and finally, where the blood count shows an increase of the leucocytes

"If the swelling has developed rapidly and attained a considerable size within a few months; if it forms a rounded prominence, covered by a marked venous network, and especially if its surface has become ulcerated and gives rise to free hemorrhages from time



Fig 833—Exophthalmic goiter

to time, one should think of *sarcoma*, which may originate in any of the structures constituting the neck and even the submaxillary gland, but which, as a rule, starts in the lymph-nodes, or results merely from transformation of a lymphadenoma, in which event it is termed *lymphosarcoma*." (Rochard and Demoulin, "*Manuel de diagnostic chirurgical*").

2. Mention should be made of the ossifluent abscesses (abscesses from congestion with breaking down of bone tissue), the existence of which is shown by the fluctuating character of the swelling with slight reducibility (liquid tumor), the fact that

motion of the head generally causes pain, and examination of the spine, which reveals either a bony deformity or the presence of a well localized point of tenderness.

3. **Lipoma**, including the *supraclavicular pseudolipoma of the emphysematous* and *adenolipoma*, probably comes next after glandular swellings in frequency A mere mention of lipoma is here sufficient. An illustration of typical adenolipomatosis is presented herewith (Fig 831).

4. Finally, there are the **vascular enlargements**, or more specifically, **aneurysm**, which is, however, an uncommon disorder, even though the supraclaviculocarotid region is a point of election for it. Aneurysms present four pathognomonic signs · They are of *fluid consistency, reducible*, and *pulsating*, whether their beats, which synchronize with those of the pulse, be visible or merely palpable; lastly, they are the seat of a *systolic blowing bruit* which is audible on auscultation. These are the well-known signs of arterial aneurysms. Arteriovenous aneurysms are always consequent upon traumatism, *e.g.*, a bullet or stab wound; the bruit is continuous with reduplication, and palpation yields the so-called "thrill," a species of fremitus with periodic accentuation

It is the custom to explain how to differentiate aneurysm from vascular goiter. A single sign is ordinarily sufficient for the purpose A goiter, even when vascular, ascends with the larynx during deglutition while an aneurysm remains motionless.

Recognition of the site of an aneurysm is of marked importance as regards operative intervention

**Carotid aneurysm** is located in the sternomastoid region The bruit is transmitted in the cervical region Only the temporal pulse is interfered with

**Subclavian aneurysm** is located in the infraclavicular region, at a point lateral to the sternomastoid The enlargement is elongated in the transverse direction The bruit is transmitted toward the axilla. The radial pulse is interfered with.

**Aneurysm of the innominate artery and aortic arch** is located in the suprasternal region, between the sternomastoid muscles. Both the radial and temporal pulses are interfered with The bruit is transmitted toward both the neck and the axilla

5. **Sebaceous cysts,** uncommon in this region, contained within the skin, painless, and of firm consistency, could hardly be mistaken for anything except a small *encapsulated lipoma,* and the untoward result of such a mistake would be slight

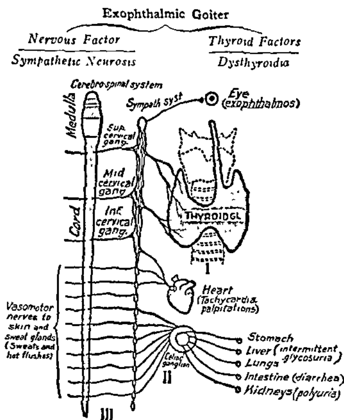



Fig. 834—Graves's disease Exophthalmic goiter. Symptoms and pathogenesis

**Enlargements in the Median Region of the Neck.**

The suprahyoid region being rapidly dealt with by simple enumeration of *adenolipoma* and *suprahyoid glandular swellings* (syphilis, lymphatic hypertrophy, or neoplasm), there remains:

The infrahyoid region, in which, the exceptional eventualities of *cysts, tuberculous lymphadenitis,* and *cancer of the larynx* (disturbances of voice production and deglutition, blood-stained and mucopurulent salivation, and laryngoscopy) having been likewise—and readily—eliminated, the remaining condition is:

Enlargement of the thyroid gland, situated definitely below the larynx, generally bilateral, and moving upward upon swallowing owing to the fact that the gland is attached to the laryngotracheal canal.

Congestion of the thyroid with temporary swelling of the neck may, as is well known, be witnessed in *pregnancy,* after *prolonged exertion,* and in the course of *infectious diseases* (small-pox, typhoid fever, eruptive fevers, etc.). It is essentially a transient condition, as is true likewise of *thyroiditis,* but it may at times be associated with the symptomatic picture of mild and fugacious Graves's disease.

True, lasting enlargements of the thyroid (cysts and solid enlargements) are, from the medical aspect, dominated by the question of *Graves's disease* and *dysthyroidia.* The problem which arises, and which is most important to the physician, is this:

*Is the condition* a simple goiter (solid or cystic) without appreciable disturbance of gland function—without dysthyroidia?

*Or is it* a goiter with overfunctioning of the thyroid or hyperthyroidia (exophthalmic goiter) or with reduced function or hypothyroidia (myxedema)?

Briefly, in every goiter case, of whatever degree and variety, one should look carefully for the signs of hyperthyroidia given in the subjoined table, which covers the main clinical features and probable pathogenesis of the disturbances of thyroid function.

* * *

Mention may here appropriately be made of the profound disturbances of metabolism which generally accompany exophthalmic goiter and of which the metabolism of glucose serves as a rather satisfactory index In this connection one may also refer to the possibility of the simultaneous presence of exophthalmic goiter and diabetes, as emphasized by Marcel Labbé, and the frequency of intermittent glycosuria and of alimentary glycosuria. Hyperglycemia is the rule in Graves's disease. American writers (Goetsch and Lueders) have, moreover, called attention to the fact that intramuscular injection of 0.5 c.c. of 1:1000 adrenalin solution in hyperthyroid subjects causes not only a marked pulse acceleration and rise of blood-pressure but also, as a rule, in the succeeding two hours, a varying degree of glycosuria.

Lastly, we may recall the relative frequency of the *signs of incipient Graves's disease in the tuberculous.* It seems probable that the manifestations are induced by the tubercle toxin, acting selectively on the vegetative nervous system—just as in "complete" Graves's disease such excitation is brought about by an endocrin hypersecretion.

**Chief Clinical Signs of Exophthalmic Goiter.**

1. **Enlargement of the thyroid gland** (goiter)
2. **Exophthalmos** and the associated ocular signs· Staring expression (Stellwag), failure of upper lid to move with the eyeball in looking downward (von Graefe); deficient convergence (Mobius)
3. **Permanent tachycardia,** respiratory arhythmia, palpitations, vasomotor manifestations (transient erythema, dermographia), sphygmolability, and febricula.
4. **Static tremor,** exaggerated reflexes, nervousness, subjective sensation of heat, and various spasmodic disturbances (asthmatoid attacks, gastrospasm, spastic constipation, etc )
5. **Profuse sweats,** unusual salivary secretion, and tendency to diarrhea on slight provocation
6. Polyuria, frequent urination, and intermittent glycosuria.
7. Sexual perturbations.

**Chief Clinical Signs of Hypothyroidia.**

1. **Atrophy and sclerosis of the thyroid,** possibly, **though rarely, in** conjunction with apparent hypertrophy, or, more frequently, with a doughy condition of the tissues of the neck
2. **Doughy condition of the tissues,** thickening of the integument, with a swollen, waxy, myxedematous appearance (false edema, firm and resilient)
3. **Physical torpor,** sluggishness, inertia, slowness of the movements, and sensitiveness to cold
4. **Mental torpor,** apathy, tendency to backwardness, and slowness of mental reactions
5. Diminution of the sweat and sebaceous secretions; skin dry and scaly Impaired nutrition of the skin and hairy covering.
6. Reduced urinary output (oliguria).
7. Sexual anorexia.

# NERVOUSNESS.

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A definition of the term "nervousness" is badly needed for the very reason that it is so commonly used. What is the meaning of the term "being nervous?" One may be nervous in a variety of different ways, and the mere attempt to have patients who describe themselves as being "nervous" give a definite account of their symptoms (often an impossible task) is enough to show that this term, as commonly used, is possessed of either vague or variable meanings which make it unsuitable for legitimate clinical use.

Unquestionably the proper thing to do is to decompose *"nervousness"* into its several primary factors and to make a systematic search, in a patient considered "nervous," for the typical stigmata and symptoms, both motor, sensory and special sense, mental, visceral and vasomotor

I **Motor nervousness** is manifested especially in *restlessness* and *exaggeration of motor responses* In one group of cases the patient is shifty, restless, unable to keep still, and actually has "the fidgets." Such motor excitement is, as is well known, practically a normal condition in children In the adult it is often associated with mental excitement, insomnia, anxious states, etc It is one of the attributes of the emotive or nervous constitution.

In a second group of cases, the patient exhibits uncontrollable contractions of varying frequency in some portion of the body: Twitchings, contractures, tics, or tremor (see *Tremor*).

This form of motor nervousness is met with in *chorea* and the *choreiform states*, in *tics*, and in many neurotic disturbances. A particular search should be made for

Somatic causes, foremost among which are:

1. Intoxications, principally *alcoholism*, less frequently cocainomania, opium habit, lead poisoning, etc

2 *Hyperthyroidia*. Graves's disease, exophthalmic goiter, incomplete (*fruste*) Graves's disease, and, in a general way, over-

activity of the internal secretions—thyroid and ovarian or testicular, and hypercrinia.

**Mental causes:** Strong emotional impressions, overwork, and passional states inducing psychoneurotic disturbances. Such motor nervousness is often the external expression of a deep mental injury. The patient has some poignant burden in his heart; he rebels, and the pain experienced induces lasting nervousness A historic example of this is that of the wandering, ill-used Empress of Austria, Elizabeth.

**II. General sensory and special sense nervousness** is manifested in an *hyperesthesia*, either *general* or selective, toward external stimuli,—whether tactile, gustatory, auditory, olfactory, visual, or cutaneous impressions be felt to an excessive or actually painful degree, or even awaken distressful motor or visceral reflexes.

The disturbance is especially evident as regards the auditory function. The subject is startled by the least sound, jumps when a door is opened, and complains of hyperacusis. Frequently such a patient is, moreover, unable to stand bright illumination or direct sunlight (as exemplified in the obstinate facial neuralgia sometimes experienced on the Southern Coast of France) At times olfactory hyperesthesia is manifest in that the subject is able to perceive odors which persons considered normal cannot smell, yet which analytic procedures show to be actually present Not rarely one observes in these cases a cutaneous hyperesthesia, demonstrated by pricking with a pin or by mere grazing of the skin surface, as well as a heightening of the tendon and skin reflexes.

This "general sensory and special sense nervousness" is frequently accompanied by neuralgia of varying kinds There is thus present an actual diathesis—an actual *algic, hyperesthesic constitution.*

Special mention should be made of the "general sensory and special sense nervousness" manifest in a "meteorologic (cosmic) hyperesthesia" These patients are actual meteorologic esthesiometers or, as they frequently describe themselves, "regular barometers" They are oversensitive to variations of temperature, of barometric pressure, and of humidity, to electro-magnetic

variations, and unquestionably also to many other cosmic variations as yet unknown to us. Some forecast and feel changes in the weather, sharp changes of barometric pressure, shifting of the direction of the wind, the precipitation of mist, etc., with extreme accuracy and under such conditions as exclude all possibility of fraud. The author has often had occasion to witness a rheumatic, neuralgic patient, sitting in the evening in a closed room with the shutters drawn and curtains pulled together, shut off from any direct or indirect contact with the outer air, announce with infallible precision a rise or fall of barometric pressure of moderate extent (5 to 10 millimeters) or a shift in the direction of the wind or the onset of snowfall, etc. Every one may readily observe phenomena of this type among his associates The events are such as would suggest that the majority of meteorologic changes cause a disturbance of balance of the body fluids, objectively manifested in various physical effects and subjectively in pain.

Whatever explanation be conceived as to the intimate causation of these disturbances, it is hard not to think of them as being dependent upon an unusually unstable electrochemical balance in the body fluids, a species of supersaturated state on the threshold of precipitation and in relation to which the least shock or perturbation suffices to bring on an intra-organic reaction involving the precipitation of some noxious agent either at the nerve-terminals (*neuralgia*), the joints (*arthralgia*), or certain internal organs (*visceralgia*) In this way may be roughly described the relationships existing between the as yet rather poorly defined groups of patients with "a tendency to" rheumatism or neuralgia, at present loosely combined under the term "neuro-arthritism"

III Psychic nervousness is perhaps a commoner condition Its outstanding feature, on the whole, is mental instability in all its different expressions. The subject is emotional, and the least untoward event causes him to lose his self-possession, he is not "master of his own acts" Often periods of enthusiastic excitement and exaggerated optimism alternate with periods of depression and unwarranted pessimism

In the condition characterized by excitement the subject is

irritable and even impulsive; he quickly becomes angry and even violent

In the depressed condition, he is morose, anxious, aboulic, and is readily seized with apprehension or actual fear

The question of the "neuroses" and "psychoneuroses" might appropriately be taken up here. We shall not do so, however, merely reminding the reader that in these cases the chief aim of the practitioner should, seemingly, be to ascertain:

1. Whether the existing psychopathic disorder is not the expression of a concrete somatic condition, the most important disease in this connection being general paralysis, as well as some common, latent or overlooked morbid state such as diabetes, arteriosclerosis, intoxication (as by alcohol, etc ), tuberculosis in its insidious stage, malaria, etc.

2 Whether the psychopathic disorder is not the necessary and perhaps inevitable consequence of some conscious mental stress, such as secret sorrow, frequently present in young girls and women, or professional difficulties or disappointments, which are responsible for a high percentage of embittered, anxious, nervous individuals, etc Political, literary, and even medical pursuits yield a host of such cases

3 Whether the psychic nervousness is not, properly speaking, a constitutional, congenital, and frequently inherited state Nothing could be more illuminating in this connection than the simultaneous examination of a mother and her daughter, the latter showing the same psychopathic manifestations as the former, but with twofold or even threefold intensity.

The highly suggestive psychologic conception of hysteria considered as a disorder characterized essentially by a contraction of the "field of consciousness" which allows the subject to group together at a given moment only an extremely restricted number of sensations and recollections accounts satisfactorily for this variety of "psychic nervousness," with its expressions in the form of instability, impulsiveness, psychic and moral incoordination, suggestibility, etc, a single idea, image, or sensation sufficing to overcrowd the field of consciousness, so that one idea drives out another.

**IV. Visceral and vasomotor nervousness.**—The *"spasmodic"* and *"paretic"* manifestations predominate in this group, whether the spasmophilia be chiefly visceral, and capable of causing very various phenomena such as spasm of the esophagus, meteorism, regurgitation of food, dyspepsia, constipation, retention of urine, etc., or cardiovascular, being represented by the anginal symptom-group, neurocardiac erethism, the tachycardiac neurosis, spasm of the vessels, hyperidrosis, dermographism, acrocyanosis, disturbances of skin temperature, etc. In these cases *sympathetic disturbances* predominate

In some subjects there is observed an actual vasomotor ataxia characterized by evidences either of vasodilation or vasoconstriction or of a mixture of these two varieties of vascular disorder. Graves's disease represents the extreme vasodilator type, and Raynaud's disease, the extreme vasoconstrictor type. Between these two are found many intervening varieties, of varying intensity and location, such as simple urticaria, dermographism, nervous edema, drug idiosyncrasies, hay fever, intermittent albuminuria, a tendency to purpura and petechiæ, multiple angiomata of the skin, and paroxysmal tachycardia. The disturbances at the menopause present essentially this condition of vasomotor ataxia.

**Clinical tests.**—There are available, indeed, certain elementary clinical tests which enable the practitioner to detect such excessive irritability of the sympathetic system

(*a*) The *vasomotor skin reactions induced by rubbing or by the application of heat or cold* When exaggerated, these lead to *dermographism,* as in the "meningeal line", when perverted, to reactions in the opposite sense, *viz*, redness after the application of cold and pallor following the application of heat.

(*b*) The *reactions as to heart frequency induced by the respiration.* Normally, the influence of the respiration on the pulse is practically *nil* Under abnormal conditions, however, the pulse becomes irregular and arhythmic during respiration; this constitutes the so-called "respiratory arhythmia" met with in young subjects and many neuropathic patients.

(*c*) The *trigemino-cardiac reactions,* the one most investigated so far being the oculocardiac reflex Normally, slowing of the

pulse rate results from pressure upon the eyeballs. Under abnormal conditions, the pulse-rate is unchanged or even accelerated.

These tests are of exceedingly great importance in the frequently difficult diagnosis of the *cardiac neuroses* (tachycardiac neuroses).

Whether present singly or in combination, the various nervous symptoms above referred to, sometimes associated with insomnia, paretic or convulsive disturbances, headache, asthenia, mental clouding, various disorders of the tendon- and skin- reflexes, and sometimes even states of mental confusion with amnesia, delirium, and hallucinatory restlessness, constitute the main factors in the *neuroses* and *psychoneuroses*—hysteria, neurasthenia, Krishaber's cerebrocardiac neuropathy, Dupré's emotive constitution, the anxiety neurosis, cardiac neurosis, and Grasset's psychosplanchnic neuropathy. Sometimes the clinical symptom-groups given in text-books as characteristic of these various neuroses and psychoneuroses are sufficiently well marked and differentiated as to permit of actually applying to the case a fairly precise designation, such as *neurasthenia* (with its stigmata: type of special headache, amyosthenia, brain depression, etc ); *hysteria* (with its characteristic pithiatism); *neuropathy* (with its stigmata: special type of headache, amyosthenia, brain depression, etc.); *psychosplanchnic neuropathy* (with its debility of the higher mental processes, hyperkinesthesia, and psychosplanchnic interdependence); the *emotive constitution*, and the *anxiety neurosis*, but the symptoms of these disorders are dovetailed and superimposed, with the result that differentiation is often a very difficult task.

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Somewhat recent investigations have drawn the attention of the profession to two types of nervousness—which, as a matter of fact, overlap—the **emotive or emotional constitution** and the **anxiety neurosis.** Both of these are of marked clinical importance; hence it is deemed advisable to present a short account of each, the first as described by Dupré, and the second, by Heckel

## I.—THE EMOTIVE CONSTITUTION.

Dupré[1] proposed some years ago to set apart, under the appellation *emotive constitution*, a special type of imbalance of the

[1] DUPRÉ. *Acad. de méd*, Apr. 2, 1918.

nervous system, characterized by diffuse erethism of general, special sense, and psychic sensitiveness; by inadequacy of motor inhibition, reflex as well as voluntary, and clinically manifested by responses abnormal in degree, diffusion, duration, and lack of proportion to their exciting causes

Hyperemotivity, a normal condition in infants and very frequently present in older children (infantile nervousness) disappears in the adult owing to the gradual development of the inhibitory nerve paths, which insure balance and stability of the nervous system. Abnormal emotivity, usually constitutionally inherent and inherited, may be an acquired state and be the result of infectious, toxic, and especially traumatic factors, such as intense or repeated body commotions or emotions.

Emotion, indeed, often sensitizes the nervous system to subsequent emotional stresses, and through a species of *emotive anaphylaxis* may bring about a constitutional emotivity. In contrast to such cases one may, on the other hand, observe in well balanced subjects a gradual habituation to an entire group of emotions, a rather remarkable *emotive immunity* being thus conferred through repetition of like emotional stresses

The emotive constitution is characterized by two groups of permanent signs, the physical and the mental

**Physical signs.**—*Exaggerated reflex action,* diffuse and involving both the tendon, skin, and pupillary reflexes. Sensory *hyperesthesia,* with sharp, extensive, and protracted motor responses, principally as regards gestures and speech. *Loss of motor equilibrium,* manifested in spasm of viscera, as in pharyngo-esophageal spasm, gastroenterospasm, bladder spasm with frequent urination, and palpitations. Emotive tremor in all its numerous expressions, such as tremor of the extremities, shuddering, quivering, shivering, chattering of the teeth, stammering, transient myoclonic movements, tics, etc *Functional inhibition* and loss of power of motor structures, of a transient nature, as in sudden giving way of the knees, inability to speak, or relaxation of the sphincters. *Loss of circulatory equilibrium,* manifested in tachycardia, sometimes occasional but often permanent and paroxysmal; instability of the pulse Alternate peripheral vasoconstriction and vasodilation; dermographism. The relationship of these circulatory disturb-

pulse rate results from pressure upon the eyeballs. Under abnormal conditions, the pulse-rate is unchanged or even accelerated.

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normally as physiologic, functional manifestations. The former, *anxiety*, characterized by disquiet, restlessness, and mental disturbance of varying degree, is produced normally and casually under the influence of emotional stresses, fear, and under all circumstances in which life and the preservation of the individual are menaced. It is ordinarily accompanied by certain concomitant manifestations, physical, organic, or somatic. It is these concomitant manifestations which characterize *angor, i.e.*, a distressful sensation of constriction (from the Greek word, ἄγχειν, to choke) involving various systems of the body. We thus have:

(*a*) *Respiratory angor*, consisting of a feeling of thoracic discomfort, of pressure on the sternum, of tightening of the inner and outer musculature of the chest, and of tightening of the bronchi; whence, oppression, fear of asphyxiation, terror, respiratory spasms, sighing, aphonia, and cough—all of emotive and anxious origin.

(*b*) *Cardiovascular angor*, simulating angina pectoris in all its grades of intensity (emotive or anxious pseudoangina pectoris), manifested in sensations of gripping at the heart, of constricted heart, or of unduly large heart, by subjectively noticeable heartbeats, palpitation, arhythmia, faintness preceded by vertigo, vascular phenomena, resulting in coldness of the extremities, beating arteries, and flushing or pallor of the face or body.

(*c*) *Digestive angor*, characterized by interference with swallowing, the emotive "globus," epigastric discomfort with a feeling of weight on the stomach, anxious gastralgia with or without pyrosis, nausea and even vomiting, colicky pains, tenesmus, constipation or diarrhea, arrest or excess of bile secretion, white or unduly colored stools, and cholemia or sometimes jaundice.

(*d*) *Cerebral angor*, associated with a feeling of constriction of the temples, of narrowing of the skull, and of emptiness in the head with consequent inability to maintain thought. There is also a typical facial expression. The facies of anxious emotivity is characterized by a transversely wrinkled forehead with two vertical folds due to the muscles of the supraorbital ridges and by accentuation of the nasolabial folds, descent of the labial and palpebral commissures, and sometimes, in the presence of marked terror, protrusion of the eyeballs. The expression of preoccupied con-

ances, chiefly *permanent tachycardia,* with certain types of high blood-pressure remains to be ascertained, especially in the subjects free of arteriosclerosis and renal disease *Loss of thermal equilibrium,* as shown in objective variations of temperature, detected by local thermometric tests, and subjective sensations of heat and cold, chiefly in the extremities. *Loss of glandular equilibrium,* with spontaneous or emotionally induced variations of the sweat, salivary, lacrymal, gastrointestinal, urinary, genital, and biliary secretions. *Disturbances of intervisceral reflex action,* taking place, in the major systems of the body, through association of spasmodic conditions, secretory disorders, and functional stimulation or inhibition, brought about through abnormal reflex effects exerted upon one organ by another along the vagosympathetic or cerebrospinal nerve paths

**Mental signs.**—Undue impressionability, a distraught condition, apprehensiveness, anxiety, irritability, and impulsiveness More or less constant or remittent, and frequently paroxysmal, these morbid conditions appear in alternation or combination and form a permanent basis or soil upon which appear and develop the emotive symptom-groups, timidity, scruples, doubts, obsessions, phobias, simple or delirious states of anxiety, and psychosexual emotive abnormalities. In the more severe cases there appear attacks of anxious melancholia, and chronic states of obsession with ultimate development of incurable delusions of self-accusation, hypochondria, or negation.

Constitutional emotivity, which, to be sure, is not incompatible with normal or unusual intellectual attainments and affectivity, is frequently combined with other neuro-psychopathic states, notably neurasthenia and hysteria, with which it exhibits certain interesting relationships as regards combination or sequence, but from which it should be clearly distinguished.

## II.—THE ANXIETY NEUROSIS.

The following data concerning the *semeiology of anxiety* and more particularly of the anxiety neurosis are based on Heckel's monograph on this subject[1]:

**Pathologic physiology.**—*Anxiety* and *angor* (*angoisse*) occur

[1] HECKEL: *Névrose d'angoisse,* Masson, publ, 1917

present alone, in incipient forms, cannot be specifically recognized and lead to numerous diagnostic errors

It will have been noted, then, that *anxiety is an emotive state, especially of a conscious sort, made up of mental distress, worry, and doubt, together with a sensation of physical constriction to which the term angor is particularly applied* The essential features of the latter are, in the first place, a feeling of contracture or spasmodic condition not only of the voluntary muscles but more especially of the visceral involuntary muscles, and secondly, of a distressful modification of the internal sensibility or cenesthesia

* * *

Under normal conditions anxiety follows any sort of emotional stress brought about by external or internal conflicts, to which it remains sufficient and proportionate

Under abnormal conditions the reactions from morbid emotions are excessive and unduly prolonged, whereas the causes responsible for them are futile and seemingly absent, in the latter instance, anxious emotivity or anxiety would appear, then, to be spontaneous, but this is not actually the case, as the mechanism of emotive sensibility is always set in motion by stimuli, often reflex and unconscious, some of which are of psychic and others of organic origin

In short, angor is a condition of bulbar origin, manifested in reactions of the body dependent upon disturbance of the nuclei of the vagus or, more exactly, the bulbar origin of the vagus and of the sympathetic This disturbance may reach the medulla by descent from the cerebrum where it originated in the psychic sphere—in which event the condition is a descending angor of psychic origin,—or it may ascend from the depths of the organism toward the bulbar center of angor along the visceral branches of the vagus and of the sympathetic—in which event the condition is an ascending angor of organic (visceral) origin. Often both routes of ascending, vagosympathetic transmission are simultaneously stimulated by the group of slight visceral commotions the knowledge of which as a whole by the brain constitutes cenesthesia (obscure consciousness of favor-

centration, of grief, or of fear and the preoccupied or tragic facies of the anxious patient are due to constriction of the nasal, ocular, and oral orifices by the contracted muscles, to a narrowing of the face as a whole, and to the vertical traction exerted along the facial lines and muscular furrows, as well as to the subject's shifting, worried, distracted glances and at times to dilatation of the pupils. This anxious facies is most striking under marked emotional stress or in the presence of abject fear

Lastly, cerebral angor or anxiety is associated with a psychic state expressed in manifestations which are:

1. *Mental, viz.*, worry, disordered thought, incoherence of mental association or even marked mental impairment with amnesia and delirium.

2. Such anxiety is also expressed in *motor phenomena*, such as restlessness of the body as a whole, inability to remain in one place; sudden, purposeless, contradictory, jerky, discontinuous movements, stamping on the floor; sometimes limited or exuberant gesticulations; at other times headlong flight or, on the contrary, motionlessness, stupor, and retirement; there may also be exclamations, complaints, and sobs

Motor reactions may also be manifested in spasm or contracture of smooth as well as striped muscles, local or general tremor with chattering of the teeth, and even tonic and clonic convulsions, constituting the *spasmodic attack of anxious emotivity*, which is nothing other than the well-known nervous spell formerly confounded with hysterical seizures.

3 The attack of anxiety also exhibits a *secretory manifestation* in the form of lacrymation, profuse sweats, emission of ordinary or colorless urine, salivation, and diarrhea, where there is secretory stimulation, or, on the other hand, inhibition of secretion as shown by dryness of the mouth, constipation, acholia, anorexia, apepsia, etc.

All these motor and secretory manifestations, which are an expression of the lowest ebb of anxious depression, lead to cessation of the latter through an actual process of "release"

4. Lastly, the *sensory manifestations* of anxiety are neuralgic pains along the thoracic nerve-trunks, intercostals, sciatics, trigeminals, etc., headache, and rheumatoid pains, which, when

## NEUROTONIAS.

| REFLEXES. | | ABNORMAL STATES. | |
|---|---|---|---|
| Oculocardiac, O or —.<br>Adrenalin, +.<br>acceleration,<br>tachycardia,<br>glycosuria (?)<br>etc.<br>Atropine, +.<br>General, +. | *(HYPER)*<br>SYMPATHETICO-<br>TONIA. | *General* | permanent:<br>**Graves's disease.**<br>temporary<br>**emotional,**<br>**passional,**<br>**febrile, digestive**<br>(2nd stage). |
| | | *Local* | **Neurovascular spasms.**<br>**Raynaud's disease.** |
| Oscillating,<br>sometimes +,<br>at others —. | | *Oscillating.*<br>*Cyclic.* | **Puberty.**<br>**Menopause.**<br>**Vasomotor instability.**<br>**Cyclic phases of psychoneuroses.** |
| Oculocardiac, +<br>Slowing (bradycardia).<br>Atropine, +.<br>Acceleration (tachycardia).<br>Adrenalin, slight or nil<br>Pilocarpine, +.<br>Salivation, sweating, depression<br>General, variable,<br>chiefly —. | VAGOTONIA. | *General* | **Neurasthenia.**<br>**Hypophyxia.** |
| | | *Temporary* | **Depressive psychoneuroses.**<br>Post-infectious or post emotional **shock conditions.**<br>Digestion (1st *stage*)<br>**Sleep.** |
| | | *Local* | **Asthmatoid states.**<br>Asphyxia of the extremities |

## COMPLEX CASE OF NEUROTONIA OF THE TYPE: GASTRO-ENTERO-NEUROSIS.

### SECONDARY REFLEX SYNDROMES.

Cerebral and special sense: Migraine, tinnitus, vertigo.
Respiratory: Nervous pseudo-asthma (*excitation of vagus*).
Circulatory: *a* Palpitations, arhythmias, tachycardia, angor (*excitation of aortic sympathetic*)
*b.* Cold extremities (*sympathetic angiospasm*)
*c.* Emotive erythema; vasomotor disturbances
Intestinal: Constipation, diarrhea, colicky pains, etc (syndrome of enteroneurosis).
General: Metabolism *Alimentary glycosuria*
Nervous irritability *Tremor.*
Asthenia. *Neurasthenia*

### PRIMARY INITIAL SYNDROME.

Predominant and permanent solar syndrome:

1 Paroxysmal gastralgic attacks (*neuritis of the solar plexus*).

2 Pain on aortic pressure (*periaortic plexus*).

3 Disturbances of gastric function (*hypertonia, hypersecretion, vagus excitation*).

4 Visible aortico-epigastric pulsations

able or unfavorable conditions of intimate functioning of the body organs).

Yet, while bulbar angor of psychic origin is dependent upon a brain primarily disturbed through some external emotion causing cerebral anxiety, it may also be initiated through the transmission to the brain of an organic ascending angor. In most instances, psychic cerebral angor or anxiety is so closely combined with bulbar angor of somatic and visceral origin that it is sometimes hard to distinguish the origin of these manifestations.

Anxiety and angor are symptoms of marked clinical interest from which every physician should be able to obtain diagnostic and therapeutic indications of prime importance in practice.

**Clinical conclusions.**—Heckel's investigations lead to the conclusion that the physician should, for purposes of diagnostic guidance, recognize three main classes of angor and anxious states· 1. Angor consequent upon functional disturbances or pathologic conditions of internal organs 2. Angor present in disturbances of general nutrition 3 Essential angor or the angor neurosis.

**Etiologic résumé.**—Aside from the favoring influence of heredity (Dupré's emotive constitution) and of race (Semitics and Latins of the Mediterranean division), symptomatic angor and anxiety, in common with the anxiety neurosis, may be consequent upon inherited tendencies of the metabolic type (gout, diabetes, obesity, etc ; hereditary arthritism) and upon all factors causing emotion (sentimental emotion or ungratified sexual desire) Emotional shocks, traumatism, nervous impressions, overwork, and fatigue are the usual causes of acquired angor and anxiety, certain infectious diseases or intoxications are predisposing factors. Heckel has demonstrated the relationship of tuberculosis and the metabolic diseases to angor. Casual slight causes, such as anger and changes in the weather suffice to bring on attacks in those predisposed

* * *

Let it be repeated, in conclusion, and even more earnestly than in earlier sections, that a diagnosis of *neurosis* or of *psychoneur-*

*osis,* even with a special symptom-group attached, but without etiologic qualification, is a diagnosis "by exclusion" or "makeshift" diagnosis with which one cannot rest satisfied. The physician should always make a systematic attempt to ascertain the casual, provocative or exciting cause of the "psychoneuropathic" syndrome.

The methodical examination to which the patient is subjected should, as always, be *thoroughgoing,* though dealing more particularly with the nervous system, blood-pressure, urine (always an indispensable procedure), blood (urea and Wassermann), thyroid gland, etc.

In patients below forty the practitioner should think especially of anemia, latent tuberculosis, syphilis, exophthalmic goiter, sexual excesses, and nutritional disturbances.

In patients past forty he should think especially of arteriosclerosis, diabetes, the menopause, and general paralysis.

Both before and after forty years a careful inquiry should be made for psycho-emotive causes, such as overwork, psycho-venereal excesses, and emotional shocks.[1]

* * *

The previously mentioned, relatively recent conceptions of sympathetic overaction (hypersympatheticotonia or *sympatheticotonia*) and of vagal overaction (hypervagotonia, hyperparasympatheticotonia, or *vagotonia*) are now orienting clinical researches toward a synthesis of neuro-vasculo-humoral dynamic relationships, of which a systemized sketch will be found in the annexed diagrammatic tables.

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[1] Regarding the war neuroses and psychoneuroses and the anxiety neurosis, the following works may be consulted

GRASSET: *"Les grands types cliniques des psychonévroses de guerre* (*Réunion médico-chir de la XVI^e région,* Jan 22, 1917, abstracted in *Presse méd,* Aug 22, 1917, p 495)

DUPRÉ· *Constitution émotive* (*Acad de méd,* Apr 2, 1918)

J. BABINSKI and J FROMENT *Hystérie, pithiatisme, et troubles nerveux d'ordre réflexe en neurologie de guerre* (Masson, publ, 1916)

F. HECKEL *La névrose d'angoisse* (Masson, 1917)

DEVAUX and LOGRE *Les anxieux* (Masson, 1916)

ROUSSY. *Traitement des psychonévroses de gerre* (Masson, 1918).

## HYPER-SYMPATHETICOTONIA [Hyper Type: Excessive Voltage].

**I. ELEMENTAL SYMPATHETICO-VASCULAR REACTIONS:** Angiospasm.

1. Sphygmomanometry: 1st and 2d determinations, by direct and indirect methods
2. Peri-arterial traumatisms: Sympathectomy.

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**II. RAYNAUD'S DISEASE:** Local asphyxia and symmetric gangrene of the extremities

1. **Sympathetic factor: Spasm;** "dead finger," paroxysmal coldness of the extremities.
2. **Arterial factor: Arteritis** (type: typhoid fever); **atresia** (type hyposphyxia)
3. **Venous factor: Phlebitis** (type. puerp infection), **venous stasis** (type mitral stenosis)
4. **Note·** Association and analogies Angina pectoris, Raynaud's disease, retinal ischemia.
5. **Treatment according to case**
   1. Specific
   2. Sympathectomy.
   3. Physical training

---

**III. GRAVES'S DISEASE:**

A Sympathetic neuro[illegible]

1. **Tachycardia.** [illegible]
2. **Excessive emo**[illegible] reflex irritabil[illegible]
3. **Hyperglycemia.** Increased basal metabolism
4. **Exophthalmos.** Mydriasis
5. **Additional symptoms**
   - Gastric dilatation and atony Diarrhea
   - Gastric hyperesthesia Solar plexus crises with aortico-epigastric pulsations

B **Tests**

1. **Oculocardiac** inverted (acceleration) or absent
2. **Adrenalin +:** Acceleration, hypertension, anxiety, tremor, excitement, glycosuria
3. **Thyroid products.**

*Graves's disease introduces a new problem*
Relationships of
- sympathetic
- circulation
- endocrin glands

## [HYPER]VAGOTONIA [Hypo Type. Insufficient Voltage].

**1. TYPICAL VAGO-VASCULAR REACTION:**
Vagotonic oculocardiac reflex + vagal inhibition = bradycardia

**2 CLINICAL MANIFESTATIONS: PNEUMOGASTRIC—ENTERO-CIRCULATORY.**

*a* Tendency to bronchial spasms: Asthmatic attacks and asthmatoid manifestations
*b* Gastralgia, **hypersecretion, hyperchlorhydria,** aerogastria, etc —ulcer (?).
*c* [illegible]
*d* [illegible]
*e* [illegible]
*f* [illegible]

**3. DRUG TESTS.**

*a* **Atropine** or **belladonna +** · Accelerator reaction through inhibition of vagus (action in asthma)
*b.* **Pilocarpine +** Initiation of paroxysmal attack Bradycardia, sweating, salivation, etc. **Shock.**
*c.* **Amyl nitrite +**
*d* Adrenalin, strychnine, thyroid = antagonistic + action on sympathetic

**4. COMPLEX CASES** (the commonest).
Hyposphyxia
Gastro-enteroneuroses and remote results: Headaches, pains, etc
Localized vagotonias (stomach, intestine, heart, vessels. etc )

(*b*) Concomitant functional disturbances (with or without pathologic changes) in the nervous system, digestive tract, endocrin glands, cardiovascular system, kidneys, etc.

(*c*) A rise in the adipomuscular coefficient, *i.e.*, of the ratio $\frac{\text{fat}}{\text{muscle}}$, which is normally 1:10

The prognostic significance of obesity is dependent upon the importance, progression, and seriousness of the concomitant functional disturbances.

**Obesity is a clinical syndrome and not a disease, since it is** dependent upon no single cause and has no constant pathogenesis.

There may occur a temporary or permanent obesity:

1. **From superalimentation,** through *hyperphagia* or overeating, since fat may accumulate in the tissues after excessive ingestion of food rich in proteins and carbohydrates

Through *hypolipolysis in the muscles and blood*: Insufficient exercise in sedentary or invalid persons, etc—insufficient oxygenation and anoxemia among hyposphyxics, anemics, sedentary individuals confined in rooms, etc.

2. **From disturbances in various organs:**

(*a*) *Inadequacy of the digestive functions and gastrointestinal dyspepsia.* The fats, in this event, being insufficiently or not at all converted, remain in the form of neutral fats which are less readily oxidizable and accumulate more readily in the tissues

(*b*) *Insufficiency of the endocrin glands as a group, though principally of the thyroid,* thus unequal to their rôle as oxidizing or fat-splitting glands

The rôle of *thyroid insufficiency* in obesity is unquestionable, as exemplified in the obesity of thyroidectomized animals, the obesity of the earlier stages of myxedema, and the sometimes marked loss of weight caused by thyroid administration

The *rôle of the reproductive glands* is even more obvious, as in the obesity of castrated animals (capons and hogs), the obesity of eunuchs, and the obesity associated with ovarian insufficiency (Dercum's disease, obesity of the menopause, obesity after ovariectomy, obesity of puberty in chlorotics with irregular menstruation, obesity of the early months of pregnancy, etc ).

## OBESITY. [Obesus, *stout; overnourished.*]

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Obesity is derived from the Latin word *obesus,* meaning an overnourished individual. It is a poor term, since obesity is not always due to an excessive intake of food

*Obesity is essentially characterized* by a general overgrowth of adipose tissue as compared to the other kinds of tissue (especially the muscles).

It will not be inappropriate to note that there exists a certain affinity between the processes of fat accumulation in the system and the processes of sugar accumulation, as in diabetes, or of protein accumulation, as in gout. All these metabolic changes are effected through the agency of special ferments Three oxidizing ferments or oxidases preside over the chemical transformations to which sugar, fat, and proteins are subjected If one of these is lacking or becomes insufficient, there results a disturbance of nutrition featured by the appearance of diabetes, gout, or obesity. Thus, gout, diabetes, and obesity may be ascribed to such causes as react upon the production of ferments by the internal organs and to conflicts between the ferments and the substances they transform. In truth, however, the anaërobic life of cells and the conception of the endocrin hormones and hormozones has in late years lent a considerable added complexity to the question The etiology and pathogenesis of obesity seem in some respects analogous to those of diabetes and of gout; one is led to ascribe the disorder partly to the endocrin or ductless glands and in a larger measure to the nervous system.

As Heckel has very clearly shown, *obesity* is a progressively *developing symptom-group, easily corrected in its initial stage* (minor obesity), which is due to exogenous and endogenous disturbances of fat regulation and is characterized by:

(*a*) Fatty *infiltration and degeneration,* more or *less pronounced,* of the connective and even other tissues.

*lipotrophic incoördination* (the fact is clinically certain as regards localized or symmetric hyperadiposes, and probable as regards obesity). The system is mainly constituted of a group of glandular organs, *viz*, the gastrointestinal mucosa; liver, pancreas, thyroid, reproductive glands, etc., which are charged with the task of elaborating the fats and governing their conservation (lipogenesis) and destruction (lipolysis), and overactivity or insufficiency of which, constituting a lipodystrophy, induces *obesity of glandular origin.*

This lipolytic activity, however, though devolving especially on certain organs or tissues, appears to be a functional attribute of living cells in general, so that any general disturbance of cell nutrition, while usually combined, indeed, with the above mentioned glandular disturbances, may induce obesity: *dystrophic obesity of neuroarthritic or toxic-infectious origin.*

* * *

Sometimes obesity is obvious The most casual inspection shows that the individual is too stout for his height Such a diagnosis, however, brings in too much of the subjective element. A person is obese when his weight exceeds the normal as compared to his height What is this normal weight? To find out, one may turn to the tables prepared by Quételet and by Bouchard

It is more convenient, however, to adopt the following simple, elementary rule: The normal weight of an adult equals in kilograms the number of centimeters by which his height exceeds one meter Thus, 154 centimeters = 54 kilograms; 165 centimeters = 65 kilograms; 172 centimeters = 72 kilograms

This rule and the tables mentioned are quite sufficient for clinical purposes provided the following corrections required because of individual peculiarities of physique are taken into account. Human subjects may be roughly classed in three groups

*Mediolinear* The average type, in which the transverse and vertical measurements exhibit a mean, normal ratio:

$$\frac{\text{Height}}{\text{Biaxillary diameter}} = 5.6 \text{ to } 6.$$

3. **From disturbances of the nervous system.**—This cause is obvious at least in certain localized adipose states, such as Grasset's *nervous adiposis* (Duchenne's pseudohypertrophic paralysis, symmetric lipomatosis, adiposis dolorosa or Dercum's disease, etc.).

It is probably present in certain forms of obesity associated with other nervous symptoms (herpes zoster, joint disorders, scleroderma, exophthalmic goiter, etc.).

4. **From disturbed general nutrition:**

(*a*) *Neuro-arthritic obesity,* dependent upon a chronic general nutritional disorder, usually inherited and associated or variously combined with the several morbid states termed arthritis, with gout, diabetes, lithiasis, etc. The word "neuroarthritic" is here used as an accepted makeshift, without attempting to conceal the fact that, to the author, arthritism is merely a nutritional symptom-group devoid of specificity.

(*b*) *Intoxications.*—Certain intoxications, particularly of the mild but repeated variety, lead to obesity. First place may here be given to *alcoholism,* and after it come, in the order of decreasing frequency, *arsenic, phosphorus,* and *lead.*

(*c*) *Infections.*—Those of greatest interest are the *typhoid* and *tuberculous infections.* The increase of weight, sometimes excessive, witnessed during convalescence of typhoid cases is a matter of common observation. *Tuberculous obesity*—a less exceptional condition than is generally thought—is due to three factors, *viz.,* irrational overfeeding (when it is readily curable, at least at first), neuroarthritic heredity, and the complex action of the tubercle bacillus itself or its toxins in certain forms of the disease and in certain predisposed individuals (Carnot's experimental tuberculous obesity).

If all clinical and experimental data are taken into account, it becomes necessary, broadening our earlier conceptions of the pathogenesis, to recognize that *obesity is the outward expression of the organic response to a lesion or functional disturbance at some point in the lipotrophic nutritional system.*

This lipotrophic nutritional system, which is extremely complex, is governed and coordinated by the cerebrospinal and sympathetic nervous system, which insures functional coöperation in this system; whence the possibility of a *hyperadiposis of nervous origin through*

In mediolinear subjects the rule that the weight in kilograms equals the number of centimeters over one meter is quite sufficient in practical work. Example: 169 centimeters; normal weight, 69 kilograms.

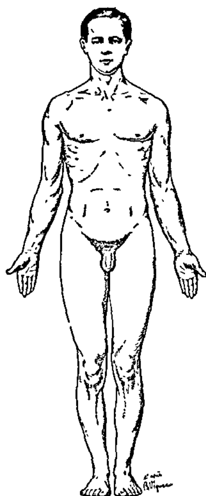


Fig 836—Mediolinear subject

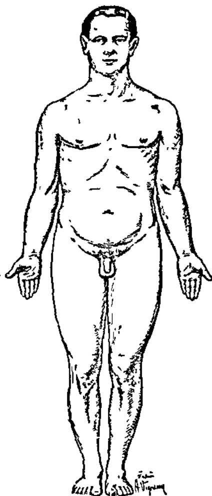


Fig. 837—Brevilinear subject.

In longilinear subjects one should allow, in calculating the normal weight, a reduction which may reach $\frac{1}{10}$. Example: 169 centimeters (longilinear), normal weight, from 69 kilograms to $69 - \frac{69}{10} = 62.1$ kilograms.

*Longilinear:* An unusually elongated type, in which the vertical measurements are manifestly excessive as compared to the transverse:

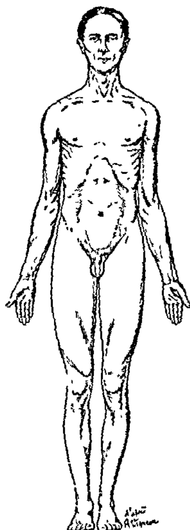


Fig. 835.—Longilinear subject.

$$\frac{\text{Height}}{\text{Biaxillary diameter}} > 6.$$

*Brevilinear:* An unusually short type, in which the transverse measurements are relatively excessive as compared to the vertical:

$$\frac{\text{Height}}{\text{Biaxillary diameter}} < 5.6$$

1. TABLE SHOWING THE AVERAGE HEIGHT AND WEIGHT AT DIFFERENT AGES (after *Quételet*) [1]

| AGE. | MALES. | | FEMALES | |
|---|---|---|---|---|
| | HEIGHT. | WEIGHT. | HEIGHT. | WEIGHT. |
| | meters | kilograms. | meters | kilograms |
| 0 year | 0.500 | 3.20 | 0.490 | 2.91 |
| 1 year | 0.698 | 9.45 | 0.690 | 8.99 |
| 2 years | 0.771 | 11.34 | 0.780 | 10.67 |
| 3 " | 0.864 | 12.47 | 0.852 | 11.79 |
| 4 " | 0.928 | 14.23 | 0.915 | 13.00 |
| 5 " | 0.988 | 15.77 | 0.974 | 14.36 |
| 6 " | 1.047 | 17.24 | 1.103 | 16.01 |
| 7 " | 1.105 | 19.10 | 1.146 | 17.54 |
| 8 " | 1.162 | 20.76 | 1.181 | 19.08 |
| 9 " | 1.219 | 22.65 | 1.195 | 21.36 |
| 10 " | 1.275 | 24.52 | 1.248 | 23.52 |
| 11 " | 1.330 | 27.10 | 1.299 | 25.65 |
| 12 " | 1.385 | 29.82 | 1.353 | 29.82 |
| 13 " | 1.439 | 34.38 | 1.403 | 32.94 |
| 14 " | 1.493 | 38.76 | 1.453 | 36.70 |
| 15 " | 1.546 | 43.62 | 1.499 | 40.39 |
| 16 " | 1.594 | 49.67 | 1.535 | 43.57 |
| 17 " | 1.634 | 52.85 | 1.555 | 47.31 |
| 18 " | 1.658 | 57.85 | 1.564 | 51.83 |
| 20 " | 1.674 | 60.06 | 1.572 | 52.28 |
| 25 " | 1.680 | 62.93 | 1.577 | 53.28 |
| 30 " | 1.684 | 63.65 | 1.579 | 54.33 |
| 40 " | 1.684 | 63.67 | 1.579 | 55.23 |
| 50 " | 1.674 | 63.46 | 1.536 | 56.16 |
| 60 " | 1.639 | 62.94 | 1.516 | 54.30 |
| 70 " | 1.623 | 59.52 | 1.514 | 51.51 |

Atonic obesity associated with polyglandular insufficiency or hyposphyxia

III. Complicated plethoric or atonic obesity (with cardiac, pulmonary, renal, or other complications)

It is true, to be sure, that the majority of these "complications" are absolutely certain to set in at some stage in the course of obesity, if the latter is not corrected in time

I Simple plethoric obesity is characterized by a simple, uncomplicated *plethora.* The plethoric is a supernormal individual all of whose functions or other features are of the "hyper" variety, but preserve a normal mutual balance. Thus, he exhibits overweight, hypertension, hyperviscosity, polyphagia, polydipsia, and

[1] One meter = 39.370 inches. One kilogram = 2.20462 pounds Avoirdupois.

In brevilinear subjects one should allow, on the other hand, an excess which may reach $\frac{1}{10}$. Example: 169 centimeters (brevilinear); normal weight, from 69 kilograms to $69 + \frac{69}{10} =$ 75.9 kilograms.

After these corrections have been deliberately introduced, if the subject's weight is distinctly above the theoretic weight, the diagnosis of obesity may be rendered.

The only features that are of actual importance from the practical standpoint are:

1. *The type of obesity* (plethoric or anemic).

2 *Its association with some allied symptom-group:*

(*a*) Gout, diabetes, or lithiasis.

(*b*) Hyposphyxia, polyglandular insufficiency, or dysthyroidia.

(*c*) Asthma, emphysema, or hay fever

3. *The presence of some complication* which, as a matter of fact, is a natural consequence in the course of the disorder.

(*a*) Cardiac or cardiovascular

(*b*) Pulmonary or cardiopulmonary.

(*c*) *Renal or vasculorenal*

(*d*) Hepatic.

These are the data upon which the treatment of the case depends

**Clinical classification.**—From the purely clinical standpoint the following classification appears to be the best because it automatically specifies the therapeutic indications—which is, after all, the essential point. Obese subjects may be divided into two diametrically opposed groups.

*Florid, plethoric, full-blooded obese subjects.*

*Atonic, asthenic, anemic obese subjects*

In either of these two groups, obesity may exist in the pure state, *i e*, without any associated morbid state or complication, or it may occur in combination with the morbid syndromes affiliated with it, or with complications Thus, with all possibilities taken into account, the following classification is offered the clinician:

I. Simple plethoric obesity.

Plethoric obesity associated with gout, diabetes, lithiasis, etc.

II. Simple atonic obesity.

combined with *polyglandular insufficiency* and *hyposphyxia*, for evidences of which a systematic search should always be made (goiter, genital dystrophies, myxedema, etc, on the one hand; low blood-pressure, high viscosity, lividity, cryesthesia, and dyspnea on exertion, on the other). *Highly serviceable indications concerning the* pathogenesis and hence also as to the treatment are thus obtained

III. **Cardiac, renal, and pulmonary complications**—Either one of the foregoing types of *obesity* may, and frequently does, become complicated with other disorders. The *heart, kidneys,* and *lungs* are the organs most frequently affected, and investigation of these organs is therefore particularly necessary in all obese patients.

(*a*) Cardiorenal Disturbances.—Subjects with *plethoric obesity* are, as already pointed out, likely victims of *cardio-arterio-renal fibrosis* (arteriosclerosis and interstitial nephritis), which is detected through blood-pressure determinations (hypertension) and examination of the urine (albuminuria and lowering of the hydruric coefficient $\frac{H}{P}$) and the heart (hypertrophy, gallop rhythm).

*Atonic obese* cases are predisposed to venous stasis (varicose veins and edema), to dilatation of the heart and cardiac insufficiency (tachycardia, low blood-pressure, dyspnea on exertion, cyanosis, and reduced urinary output), and to chlorine retention.

Both types terminate in *heart failure* and *uremia* the result of progressive cardiorenal inadequacy.

Heckel, in his book on obesity, was the first to lay stress on the marked frequency of the chronic nephritides among the obese, stating that "nearly all the obese are incipient uremics" Marcel Labbé reached the following conclusions· "The ratio of interstitial nephritis among the obese is 22 per cent before the age of fifty years and 77 per cent. after fifty years This means that in all old obese subjects renal sclerosis is almost inevitable." The author of the present work has demonstrated the above-mentioned relationship of the plethora of obese cases to chronic hydremic (hypertensive) and azotemic (uremigenous) nephritis, and has formulated rules, based on observation of the blood-pressure, blood viscosity, and daily output of urine which enable the physician to discern the moment when patients are passing from the stage of simple plethora to that of chronic nephritis (see *High blood-pressure*).

polyuria. He is a florid-complexioned, full-blooded, obese subject whose digestive, nutritive, circulatory, urinary, and other functions are of an exaggerated type but nevertheless very satisfactorily carried out; bodily vigor and the mental faculties are unimpaired

It cannot be too often repeated, however, that such a subject is predisposed to the so-called neuro-arthritic disorders, *viz.*, gout, diabetes, lithiasis, etc., and a likely victim of connective tissue deposit in the viscera, especially the arteries and kidneys

This fact should be duly borne in mind when the diagnosis is made, and the necessary precautions taken, even in the "minor obesity" stressed by Heckel, for, as this observer correctly writes "It is ridiculous to wait, in order to obtain a suitable label, until a person is deformed with fatty accumulations before warning him of the risk he is running."

Overnutrition and an arthritic family tendency are generally met with as etiologic factors in these cases

*Plethoric obesity* is very frequently associated with *gout, diabetes,* and *lithiasis,* the pathogenesis of which seems to be closely related to plethora All plethoric obese subjects should therefore be gone over carefully in this connection, being examined for evidences of gout in the joints or internal organs, of diabetes (glycosuria), and of lithiasis (high specific gravity, acidity, and uric acid content of the urine), and warned against these possible complications

II. **Anemic, atonic, asthenic obesity,** on the other hand, is manifested in a pale complexion and at times a waxy appearance; the appetite is often poor, the digestive functions are impaired, constipation is frequent, bodily vigor is below normal, indolence and apathy are habitual, the circulation is deficient, hyposphyxia frequent, and the elimination of chlorides often disturbed All test features are of the "hypo" variety, *viz.*, hypotension, hypopepsia, etc., with the exception of the body weight and viscosity, which are increased.

In these cases the etiologic factors most often met with are dyspepsia, anoxemia, intoxications, infections, and degeneration of glandular organs, such as the thyroid and testicles. Typhoid and tuberculous obesity belong in this category.

*Anemic, atonic obesity,* usually attended with slowed and vitiated nutritive processes and circulation, is very frequently, if not always,

2 Height, Weight, Average Anthropometric Segments, Fat per Segment, Fat in the Whole Body (after *Bouchard*), *continued*

| Height in Centimeters H | Weight in Kilograms W. | Weight of the Segment $\frac{W}{H}$. | Fat per Segment in Grams. | Fat in the Whole Body. |
|---|---|---|---|---|
| 149 | 53 45 | 3 59 | 466 | 6 948 |
| 150 | 54 32 | 3 62 | 471 | 7 062 |
| 151 | 55.21 | 3 66 | 475 | 7 173 |
| 152 | 56 09 | 3 69 | 480 | 7 292 |
| 153 | 56 93 | 3 72 | 483 | 7 401 |
| 154 | 57 78 | 3 75 | 488 | 7 511 |
| 155 | 58 64 | 3 78 | 492 | 7 623 |
| 156 | 59 50 | 3.81 | 496 | 7 735 |
| 157 | 60 38 | 3 85 | 500 | 7 849 |
| 158 | 61.26 | 3.88 | 504 | 7 964 |
| 159 | 62 15 | 3 91 | 508 | 8 080 |
| 160 | 62 91 | 3 93 | 511 | 8 178 |
| 161 | 63 76 | 3 96 | 515 | 8.291 |
| 162 | 64 61 | 3 99 | 518 | 8 392 |
| *163* | *65 46* | *4 02* | *522* | *8.509* |
| 164 | 66.26 | 4 04 | 525 | 8 610 |
| 165 | 67 06 | 4 06 | 528 | 8 712 |
| 166 | 67 79 | 4 08 | 531 | 8 815 |
| 167 | 68 55 | 4 11 | 534 | 8 912 |
| 168 | 69 30 | 4 13 | 536 | 9 005 |
| 169 | 69 98 | 4 14 | 538 | 9 092 |
| 170 | 70 69 | 4 16 | 541 | 9 197 |
| 171 | 71 38 | 4 17 | 543 | 9.285 |
| 172 | 72 07 | 4 19 | 545 | 9 374 |
| 173 | 72 78 | 4 21 | 547 | 9 463 |
| 174 | 73 48 | 4 22 | 549 | 9 552 |
| 175 | 74 11 | 4.24 | 551 | 9 642 |
| 176 | 74 77 | 4 25 | 552 | 9 715 |
| 177 | 75 40 | 4.26 | 554 | 9 806 |
| 178 | 76 04 | 4 27 | 555 | 9 879 |
| 179 | 76 77 | 4 29 | 558 | 9 988 |
| 180 | 77 42 | 4 30 | 559 | 10 062 |
| 181 | 78 08 | 4 31 | 561 | 10 150 |
| *182* | *78 73* | *4 33* | *562* | *10.228* |
| 183 | 79 40 | 4 34 | 564 | 10 321 |
| 184 | 80 06 | 4 35 | 566 | 10 414 |
| 185 | 80 73 | 4 36 | 567 | 10 489 |
| 186 | 81 39 | 4 38 | 569 | 10 583 |
| 187 | 82 07 | 4 39 | 571 | 10 678 |
| 188 | 82 76 | 4 40 | 572 | 10 759 |
| 189 | 83 43 | 4 41 | 574 | 10 849 |
| 190 | 84 11 | 4 43 | 576 | 10 944 |
| 191 | 84 79 | 4 44 | 577 | 11.021 |
| 192 | 85 48 | 4 45 | 579 | 11 117 |
| 193 | 86 17 | 4 47 | 581 | 11 213 |
| 194 | 86 85 | 4 48 | 582 | 11 291 |
| 195 | 87 48 | 4 49 | 583 | 11 372 |
| 196 | 88 08 | 4 49 | 584 | *11 446* |
| 197 | 88 81 | 4 51 | 586 | 11 544 |
| 198 | 89 32 | 4 51 | 586 | 11 603 |
| 199 | 89 87 | 4 52 | 587 | 11 681 |
| 200 | 90 40 | 4 52 | 588 | 11 752 |

(*b*) PULMONARY DISTURBANCES.—Obese persons are exposed to numerous complications:

*Respiratory insufficiency, anoxemia, asthma, and emphysema* —almost constant features at a certain stage of obesity.

Active hyperemia, congestive attacks, and acute edema of the lungs in plethorics, and more especially in the cardiorenal stage

Passive hyperemia, edema of the bases of the lungs, and hydrothorax in the atonic cases and more especially in the stage of cardiac insufficiency. In this group, as a matter of fact, a cardiopulmonary trend of the disease is much commoner than cardiorenal complication.

This diagnosis of the complications of obesity constitutes, on the whole, in conjunction with the causal diagnosis, the essential feature in the diagnosis of obesity, and one should not wait, to render it, until irreparable tissue lesions have set in. To wait for the appearance of gallop rhythm before rendering a diagnosis of interstitial nephritis is like waiting for the police before cutting down a man who has just been hung. As Heckel writes: "It is not the amount of fat accumulated which allows of the prognosis being made, but the intensity of the functional disturbances accompanying the obesity, whether the latter be of the major or minor variety."[1]

2 HEIGHT, WEIGHT, AVERAGE ANTHROPOMETRIC SEGMENTS, FAT PER SEGMENT, FAT IN THE WHOLE BODY (after *Bouchard*).[2]

| HEIGHT IN CENTIMETERS H | WEIGHT IN KILOGRAMS W. | WEIGHT OF THE SEGMENT $\frac{W.}{H}$ | FAT PER SEGMENT IN GRAMS | FAT IN THE WHOLE BODY. |
|---|---|---|---|---|
| 140 | 45 81 | 3 27 | 425 | 5 955 |
| 141 | 46 66 | 3 31 | 430 | 6 066 |
| 142 | 47 50 | 3 35 | 435 | 6 175 |
| 143 | 48 36 | 3 38 | 440 | 6 292 |
| 144 | 49.18 | 3 42 | 444 | 6 394 |
| 145 | 50 05 | 3 45 | 449 | 6 506 |
| 146 | 50 88 | 3 49 | 453 | 6 614 |
| 147 | 51 73 | 3 52 | 462 | 6 725 |
| 148 | 52 58 | 3 55 | 462 | 6 835 |

[1] For additional data on this question, see F HECKEL: *Grandes et petites obésités*, Masson, 2d Ed, 1920

[2] One hundred centimeters = 39.370 inches One kilogram = 2 20462 pounds Avoirdupois.

### TRANSIENT, TEMPORARY, ACCIDENTAL OLIGURIA.

After evacuation of an extensive collection of fluid, as in cysts, ascites, or pleurisy, it is the rule to observe a distinct reduction in the amount of urine. An actual drainage of water occurs toward the affected structures. The oliguria is less marked at the time of spontaneous reabsorption of an exudate, which process may instead be associated with a relative polyuria.

In *cholera, amebic* and *bacillary dysentery, infantile enteritis, yellow fever, cholera morbus,* etc., intense oliguria may occur on account of repeated, copious bowel movements, and the usual presence of albumin in the urine indicates that even apart from the withdrawal of water through the intestine the kidneys are more or less affected by the existing toxic-infectious disorder.

In another group of cases, oliguria appears to be the consequence of a reflex inhibition of diuresis, which may originate either in the urinary system itself or elsewhere Reference may here be made to the *reflex oliguria of renal colic,* which involves not only the diseased but also the opposite kidney, to *calculous anuria,* which passes off upon catheterization on the affected side, and to the *oliguria frequently induced by urethral and ureteral catheterization.*

*Peripheral stimuli, burns, traumatic injuries, neuroses* such as hysteria and neurasthenia, *painful conditions* (*hepatic colic*), peritonitis, appendicitis, and laparotomy may, as is well known, likewise induce a temporary oliguria, with excretion of clear urine of high specific gravity, of good concentration, and free of any abnormal constituents

In a final group of cases, oliguria appears in the presence of some infectious process, and this type of oliguria forms part, in conjunction with the temperature and the quality and rate of the pulse, of the most characteristic symptomatic triad giving information as to the course of the infection A moderate degree of oliguria, with a full, regular, and moderately accelerated pulse, and a temperature of varying height, but with good morning remissions, points to a favorable course and termination. Excessive oliguria, with a frequent, small, irregular pulse and

# OLIGURIA.

[ὀλίγος, *scanty*; οὖρον, *urine*; *diminished secretion of urine.*]

---

In a normal adult person on a normal diet, the average daily output of urine is from 1250 to 1500 cubic centimeters, the output in the daytime, from 9 A.M. to 9 P.M. (750 to 1000 cubic centimeters), being always distinctly greater than the nocturnal output, from 9 P.M. to 9 A.M. (500 to 400 cubic centimeters)

*Oliguria is present* when the amounts excreted are distinctly below the above mentioned average figures. Oliguria is said to be total, diurnal, or nocturnal according as the reduction affects the daily output or only that in the daytime or at night

* * *

Diuresis being closely and obviously dependent both upon the amount of water ingested either in beverages or in solid food and upon the various excretions other than the urine (cutaneous, intestinal, and pulmonary), many factors may, singly or in combination, induce under physiologic circumstances a definite diminution of the output of urine. Such factors should be thought of from the start, in order that they may be excluded: *Habitual restriction of fluids,* whether spontaneous or under the physician's direction (dry régime); one should always ascertain approximately the intake as well as the output of fluid; *spontaneous free sweats,* as from violent and sustained exertion or from summer temperatures (*oliguria in the summer is almost constant in normal individuals*); *free evacuations of the bowels,* spontaneous (transient diarrhea) or induced (purgation), etc.

The foregoing brief and elementary inquiry having been gone through, there remain:

1. Oliguria of a transient, temporary, accidental type.
2. Oliguria of a lasting, permanent, habitual type.

Gradually cardiac decompensation becomes more marked, and with it appear *continuous total oliguria,* permanent and paroxysmal dyspnea, edema, hypostatic congestion of viscera, cardiac insufficiency, and cardiac failure. The importance of watching the output of urine in heart disease and its value in connection with the prognosis and treatment are well known; the urine graduate serves in such cases as does the thermometer elsewhere

**Oliguria of cardiopulmonary origin.**—Whether primary or secondary, decompensation or cardiac insufficiency is sooner or later complicated with stasis in the pulmonary circulation, anoxemia, hyposphyxia, and even asphyxia; the viscosity of the blood rises in the absence of any compensatory ascent of blood-pressure, renal stasis becomes more marked on this account, and consequently, oliguria increases

As is well known, an opposite sequence of events may occur instead, some respiratory disorder, such as chronic bronchitis, emphysema, capillary bronchitis, etc, bringing on cardiac insufficiency and the syndrome of cardiopulmonary insufficiency causing oliguria.

So long as the kidneys remain uninvolved, the urine remains scanty, of high specific gravity, deeply colored, and highly concentrated.

**Oliguria of cardiorenal origin.**—Participation of the kidneys in the reduction of urinary secretion is shown by a definite, pathognomonic sign: The ratio of the absolute daily output of urine to the number of centimeters of mercury of differential or pulse pressure, as determined with the Pachon instrument, shows a reduction. *In a normal subject this ratio (of the daily output of urine, H, to the differential pressure, p) is equal to or exceeds* 250. *In a subject with interstitial or congestive renal involvement it is below 200 (law of Martinet).*

Simultaneously there are observed to appear more or less rapidly:

Either the evidences of hydremic, hypertensive nephritis—high blood-pressure, low viscosity, hydremia, gallop rhythm, hemorrhages, false polyuria with elimination of urine of low specific gravity, a slight degree of albuminuria, etc.

sustained hyperthermia with but slight or no remissions, points to the necessity of a very guarded prognosis.

Upon defervescence one observes a more or less marked outburst of polyuria, with emission of urine of high specific gravity, poor in chlorides but rich in urates, phosphates, urea, urobilin, pigments, etc., imparting to the highly colored urine, with its abundant brick dust sediment, a highly characteristic appearance.

## LASTING, HABITUAL, PERMANENT, OR AT LEAST EASILY RECURRING OLIGURIA.

Of far greater import from the standpoints of semeiology and treatment are the lasting, habitual or recurring forms of oliguria The major systems most frequently responsible are the circulatory and renal systems Oliguria is one of the cardinal evidences of cardiorenal inadequacy, and the heart and kidneys are always the organs to be investigated above all else in these cases.

**Cardiac insufficiency.**—*Reduced urinary output is, with dyspnea on exertion, one of the earliest signs of incipient cardiac inadequacy, hyposystoly, or circulatory decompensation* One cannot emphasize too strongly the following fundamental features: The output of urine is dependent upon renal permeability (and more probably, glomerular permeability) and upon the blood-pressure (especially the differential or pulse pressure). Where compensation for the cardiac or renal or cardiorenal disorder has been lost; when the cardiac fiber, no longer equal to its task, shows signs of becoming fatigued, the kidney, no longer receiving blood under the required degree of pressure, becomes oliguric, if, however, the heart is alone at fault, the urine is more markedly reduced in amount, but is of high specific gravity, well concentrated, and free of foreign constituents.

For a long time, as a matter of fact, oliguria is manifest only when the patient is in the standing posture (*orthostatic oliguria*), and is counterbalanced by the relative polyuria existing when he is recumbent (*clinostatic relative polyuria,* or *nycturia*), so that in the earliest stages, diurnal oliguria is compensated for by nocturnal polyuria, and the daily intake and outgo of fluid are in equilibrium.

fluid, of which clinostatic polyuria (nocturnal polyuria or nycturia) is, as a matter of fact, merely one variety.

* * *

**Oliguria of renal origin** is also deserving of some consideration.

The *oliguria of renal congestion,* constantly present in *acute nephritis,* is generally accompanied, at least in the more serious cases, by severe lumbago with dense, highly-colored, albuminous, and acid urine, yielding a sediment containing more or less disorganized red blood cells and exhibiting renal epithelia, granular and blood casts, etc. It may be the result of *poisoning by a drug,* typically cantharides, of an *infection,* as in beginning grippe with backache, or of an *autointoxication,* as exemplified in gout.

In *gouty nephritis,* the marked accumulation of urates along the uriniferous tubules checks diuresis and may induce renal congestion and oliguria, which may in turn progress to complete anuria

*Hyperemia of the kidneys* may find its expression *in the course of chronic renal disorders* in the appearance of a more pronounced oliguria.

* * *

A separate place in the classification should be made for **oliguria with cloudy urine,** whether the latter contains an excess of readily deposited crystalloid compounds, such as phosphates and carbophosphates, blood cells that have passed out from the vessels during acute hyperemia, or pus

*Oliguria with cloudy urine containing pus* may follow or even alternate with *cloudy polyuria* Polyuria is, as is well known, a customary reaction of the kidney to extrarenal urinary infection, as in prostatic hypertrophy, cystitis, or pyelonephritis, in these cases the urine is of low specific gravity and low in urea and chlorides. Cloudy oliguria following cloudy polyuria indicates that extension of the infectious process to the kidney itself has occurred, nephritis following pyelonephritis.

At times, in some cases of nephritis, the urine becomes cloudy owing to the passage of pus and blood cells into it; under these circumstances, either the kidney has undergone certain

Or those of uremigenous nephritis—headache, dyspnea, somnolence, excess of blood urea, etc.

Or those of hydropigenous *nephritis—edema, serous exudates*, diminished chlorides in the urine, etc.

Or the combined indications of a pan-nephritis, combining in greater or less degree the above several deficiencies of renal functioning.

In some instances—in fact, generally—this primary renal impairment, relatively well compensated for a varying period of time through compensatory cardiac hypertrophy, becomes associated with evidences of heart weakness only in a relatively late stage

In other instances the heart weakness is primary and the renal inadequacy secondary.

Present methods of determining the functional capacities of the heart and kidneys ordinarily yield sufficient information as to the comparative parts played by the two symptoms, and such information is of far more than mere academic interest, since it gives a clue to an efficient line of treatment

**Oliguria of cardiopneumorenal origin.**—In the final stage of cardiac, pulmonary, or renal disease, the disorder is no longer limited to the heart, lungs, or kidneys. A combined insufficiency of these major systems, closely united as they are through the circulation, exists. There is present a cardiopneumorenal insufficiency: Heart failure, asphyxia, and uremia are conjointly operative, each engendering and aggravating the others, and an extreme degree of oliguria is produced But however serious—and frequent—such a condition may be, it is not necessarily fatal, at least at the outset Active, energetic, and judicious treatment often insures to such patients an additional more or less prolonged lease of life.

**Oliguria of hepatic origin.**—*Hepatic obstruction* is likewise a factor in the production of lowered urinary excretion, and in different ways, *viz.*, through high pressure in the portal circulation and abdominal venous stasis, and through pressure upon the vena cava above the renal veins and passive hyperemia of the kidneys Oliguria of hepatic origin is known sometimes to show the characteristic feature termed *opsiuria*, or delayed excretion of ingested

# PAIN IN THE SIDE.

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The expression "pain in the side" is not infrequently used to designate acute pains in the region of the chest which may be compared to those induced by the impact of a pointed instrument in this vicinity This is a common symptom in many thoracic affections, yet one which may be practically pathognomonic in pleurisy and pneumonia.

For practical purposes it is convenient to group such "pains in the side" into those of visceral origin and those of parietal origin

**Thoracic pain of visceral origin** is typically referable to the *pleura* or *lung*.

**Pleurisy** is a common cause of such pain

*Acute serofibrinous pleurisy* induces, in general, a rather diffuse pain, sometimes extending over an entire half of the thorax, of variable intensity, and increased by cough, deep inspiration, sneezing, and often by motion and lying on the affected side. It interferes with the breathing, which becomes more superficial. The usual signs of pleurisy should be examined for, *viz.*, dulness, loss of fremitus, muffled breath-sounds, egophony, etc ; in the event of doubt, exploratory puncture will settle the diagnosis.

*Suppurative pleurisy*, aside from the usual signs of pyemia, including the severe constitutional disturbance and the special characteristics of the temperature curve, is sometimes marked by a more superficial location of the pain and greater sensitiveness to digital pressure Here again exploratory puncture will eliminate all uncertainty.

The advent of *pneumothorax* is generally marked by a sharp, intense, sudden pain, almost causing syncope and attended with extreme dyspnea

Pain is most violent, however, in *diaphragmatic pleurisy*, causing the patient to cry out and literally cutting short his respiration.

changes owing to the toxic effect of faulty metabolism, or the changes in it are of infectious origin. In the former event, the urine is readily rendered clear by dietetic measures; in the latter, its cloudy condition is harder to overcome.

Bacteria may harm the kidneys either directly (*in situ*), or indirectly through the action of their toxins and altered metabolism. Tuberculous infection affords many instances of this kind; outside of what is properly termed tuberculous nephritis, there are many cases in which tuberculous disease of the lungs, intestine, or even the joints leads to the appearance of the signs of nephritis with reduced output of cloudy urine—and sometimes slight hematuria—even though no tubercle bacilli can be found at the time by the most advanced methods; *tuberculosis of the kidneys* often sets in in this manner.

Later, the urine, reduced in amount, becomes increasingly cloudy through secondary development of many varieties of germs, especially the colon bacillus, in a kidney with lowered powers of resistance.

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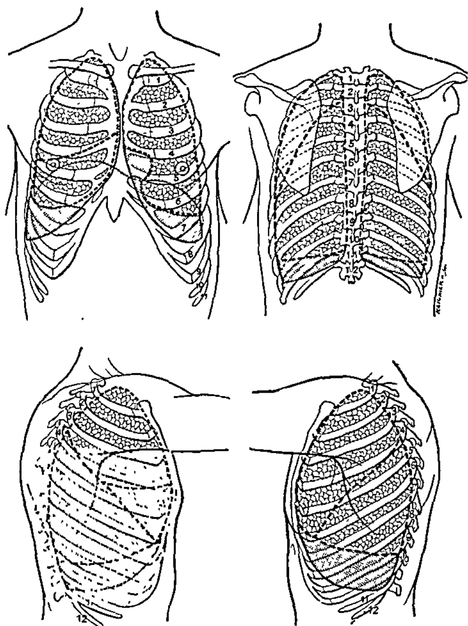
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Figs 838 to 841—Anterior, posterior, and lateral topographic features of the chest, showing the pleural culs-de-sac and the interlobar fissures.

The pain is situated lower down than in ordinary pleurisy. The five cardinal points of tenderness emphasized by Peter and Guéneau de Mussy should be investigated in these cases:

1. Between the two heads of the sternocleidomastoid

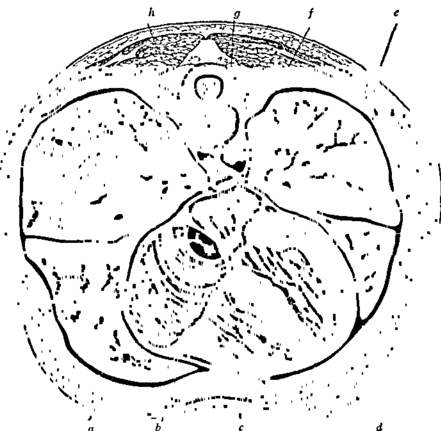



Fig 842—Relations of the heart as shown in a horizontal section through the chest of a new-born infant (*Poirier*) *a* Right auricle *b*. Left auricle *c* Right ventricle *d* Left ventricle *e* Phrenic nerve *f*. Esophagus *g* Aorta. *h* Vena azygos

2 Along the border of the sternum in the upper costal interspaces

3 The diaphragmatic point, at the junction of the sternal line (right or left sternal margin) and the line of prolongation of the bony portion of the tenth rib.

4. The insertions of the diaphragm at the base of the thorax.

5. The spinous processes of the upper cervical vertebræ

These points, which constitute, as a matter of fact, the points for stimulation of the phrenic nerve, are of service, according to Peter, not only to ascertain the condition of the diaphragmatic pleura but also that of the peritoneal covering of the diaphragm.

*Interlobar pleurisy* is marked in particular by *"fissural points"* which Sabourin describes as follows: "On either side, at the level where the third and fourth ribs mark the beginning of the chief

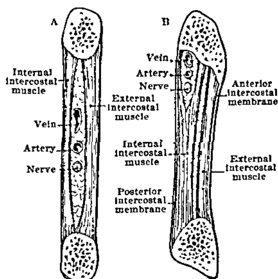



Fig 843 —Anatomic relations of the intercostal nerves.

fissure, is the *vertebral point*, at the anterior end of the chief fissure, at about the sixth rib, is the *antero-inferior point*. In addition, on the right side, owing to the differentiation of the middle lobe, there is a *post-axillary point* corresponding to the beginning of the horizontal secondary fissure and an *antero-superior point* corresponding to the axillary end of this secondary fissure. These are actually the only marginal foci of clinical interest."

At these points there is spontaneous pain, which is accentuated by motion, deep inspiration, coughing spells, etc ; pain is also induced by digital pressure.

Special mention should be made of *localized dry pleurisy in the precordial cul-de-sac*, giving rise to a precordial painful point and

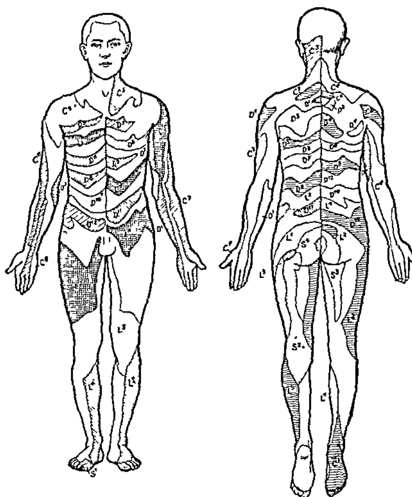
frequently attended with angor, dyspnea, and premature beats—in short, with pseudocardiac neighborhood manifestations. Careful and accurately directed auscultation elicits characteristic friction sounds corresponding with the respiratory movements; one might at times be in doubt as to the possible presence of dry pericarditis,

Figs 844 and 845—Head's zones

which may, indeed, be combined with the pleural disturbance (see *Precordial pain*).

**Inflammatory conditions of the lung** likewise induce pain in the side. Whether the disturbance present is pneumonia or tuberculosis, these inflammatory states are very often in the nature of a *pleuropulmonary "corticalitis,"* and the pleura participates, as already mentioned, in the production of the pain.

*Acute frank lobar pneumonia* represents the type of the dise
attended with pain in the side. The pain is generally more vio
and situated more anteriorly (often in the nipple line), m
"stabbing," and hence more intolerable than that of pleurisy. I



Figs 846 and 847.—Head's zones

usually accompanied with a marked chill, followed by a rapid ris
of temperature, which lead the physician to look for the usual loca
evidences of pneumonia (dulness, exaggerated fremitus, bronchia
breathing, crepitant râles, and characteristic sputum), the appear-
ance of which is, however, often postponed until much later, par-
ticularly in central pneumonia In the latter form, the disease

sometimes takes several days to reach the cortex and to become objectively noticeable through manifest auscultatory signs

As is well known, however, this "dramatic" onset is lacking in aged subjects, in whom one should arbitrarily look for the signs of pneumonia, often practically latent; the same is true, but for opposite reasons, in children.

*Congestion of the lungs* and *bronchopneumonia* may, in a varying degree, give rise to variously situated pains in the chest.

The old-fashioned notion of a "chest flux" (*fluxion de poitrine*) had to do with an inflammatory condition in the chest involving both the muscles (pleurodynia), the nerves (neuralgia), the pleura (pleurisy), and the lung (pneumonia, inflammatory congestion). While this syndrome is not specifically dealt with in our text-books, it certainly occurs clinically

An investigation of "pains in the side" is especially indicated in *pulmonary tuberculosis.* Such pains occur under two entirely different circumstances · 1. *In connection with acute pneumonic and bronchopneumonic attacks* (with especial involvement of the apex), in which they are not notably different from the pains of ordinary pneumonia and bronchopneumonia

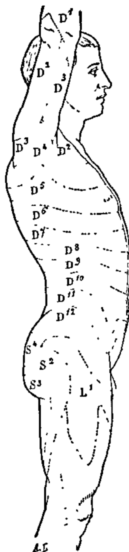



Fig 848—Segmental cutaneous distributions of the nerves of the trunk (after *Head*)

2 In a practically permanent and chronic form, in any stage of the disease, even when latent The pain is sometimes spontaneous and intermittent, but is nearly always induced or augmented by pressure upon or percussion over the supraclavicular and supraspinous regions, corresponding to the apex of the lung. It is undoubtedly

due to a *pleuropulmonary corticalitis or apical pleuritis*, which is practically a constant accompaniment of tuberculosis.

Lastly, *pneumothorax* likewise begins with a sudden, severe pain in the side, generally starting in a coughing spell or upon exertion, situated in the vicinity of the inferior angle of the scapula or the

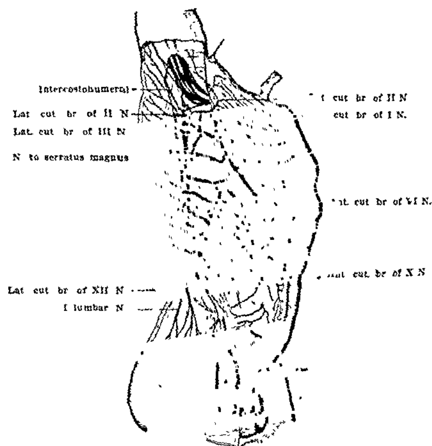



Fig 849.—Cutaneous branches of the intercostal nerves (after *Poirier*).

nipple, and accompanied by severe dyspnea with marked acceleration of the respiration. Tympany, loss of vocal fremitus, disappearance of the vesicular murmur, sometimes amphoric breathing, the coin test, metallic tinkle, and the usual history of tuberculosis or emphysema (though sometimes wholly negative in this respect), generally permit of a rapid diagnostic decision in these cases.

Various abdominal affections, especially subdiaphragmatic disorders, may give rise to pain in the side. Mention need here

only be made of the *right scapular point* in gall-stones, the *left inferior thoracic* and *precordial points* in flatulence and especially in aërophagia, and the right or left *inferior thoracic points* encountered in the different varieties of subdiaphragmatic abscess.

**Thoracic pain of parietal origin** may arise from any one of the several layers of tissue constituting the thoracic wall:

1. The **skin**, in which *herpes zoster* may occur as an outward expression of a deep-seated nervous disturbance.

2. The **muscles**, inflammation of which causes *pleurodynia*, or pains accentuated particularly by motion or palpation of the muscles concerned; pains diffuse in distribution, involving muscular rather than nervous structures, or at least exhibiting neither the characteristics nor the distribution of intercostal neuralgia.

3. The **bones**—ribs and spinal column Osteitis, osteoperiostitis (generally tuberculous or specific), or chondrosternal or costovertebral osteoarthritis, actually giving rise rather to a localized area of painful exacerbation on pressure than to a true "pain in the side." Involvements of the *spinal column* will be briefly considered below.

4. The **nerves**.—(*a*) *Intercostal neuralgia*, outlining a costal interspace with the three classic Valleix's points of hyperesthesia: 1. Posterior, just lateral to the corresponding spinous process 2 Intermediate, in the axillary line. 3 Anterior, slightly lateral to the sternum

(*b*) *Herpes zoster* (zona; shingles), referred to above as a skin manifestation, is as a matter of fact ' a neuralgia-neuritis with neuritid or nervous (nerve), radicular (ganglion), or segmental (horizontal zone) distribution, and with herpetic vesicles forming a burning half-girdle" (Grasset).

(*c*) The *pseudoneuralgia* (*neuritis*) of *Pott's disease*, of *vertebral cancer*, of *vertebral spondylitis*, of *aneurysm of the abdominal aorta*, and of *cancer cases without spinal involvement*, is generally bilateral and associated with hyperesthesia and accentuation of the pain by percussion of a definite vertebral zone

5 Lastly, in the **spinal cord**, *tabes* and *dorsal meningomyelitis* may give rise to girdle pains or to thoracic constriction with or without lightning pains, gastric crises, and the usual signs of tabes (Argyll-Robertson pupil, ataxia, astasia, loss of knee-jerks, etc.).

**Exceptional cases.**—One cannot conclude a discussion of the symptom, "pain in the side," without referring to *Head's zones of cutaneous hyperesthesia.* Head showed that in many disorders of internal organs investigation of skin sensitiveness demonstrates the existence of corresponding, well-defined hyperesthetic (hyperalgesic) zones, and on the basis of natural sequence and reciprocity, concluded that the observation of such a zone of hyperalgesia in any given case definitely means the existence of some disturbance of the underlying deep organ. His conceptions do account for many otherwise inexplicable pains and are of considerable service in the clinical study of many more or less latent affections of viscera. It seems advisable, therefore, to recall here the various associations of skin regions to the viscera of the chest and abdomen, as established by Head Through their agency the author has frequently been enabled to announce the existence of otherwise completely latent foci of inflammation in the pleura and lung.

The subject appears of sufficient practical importance to warrant the reproduction *in extenso* from Head of a complete table showing the areas of pain referred to the skin surface from visceral disease.

**Table Showing the Relationships Between the Thoracic and Abdominal Viscera, the Spinal Segments, and the Peripheral Nerves of the Trunk.** (After Head, in Poirier's "Anatomy")

[The question marks following certain pairs of nerves in the table are intended to call attention to the fact that the transmission of pain does not occur constantly in the field of distribution of these nerves]

| Organs | Nerves along which pain is referred to the parietes in visceral disease | Remarks |
| --- | --- | --- |
| Heart and aorta | $D_1$, $D_2$, $D_3$, $D_4$ | In angina pectoris the referred pain extends down the arm to the area of distribution of $D_1$, $D_2$, and $D_3$, and also in the thoracic region in the segments $D_5$, $D_6$, $D_7$, $D_8$, and $D_9$ |
| Lungs | $D_1$, $D_2$, $D_3$, $D_4$, $D_5$ | The pain in pneumonia is more especially localized in the 4th and 5th costal interspaces, collaterally the area of referred pain may extend into the segmental distributions $D_6$ and $D_7$ |
| Esophagus | $D_5$, $D_6$, $D_7$, $D_8$ | |

| ORGANS | | NERVES ALONG WHICH PAIN IS REFERRED TO THE PARIETES IN VISCERAL DISEASE | REMARKS. |
|---|---|---|---|
| Stomach | Cardiac region | $D_6$, $D_7$ | Xiphoid point and spinal point in gastric ulcer |
| | Pyloric region | $D_8$, $D_9$ | |
| Small and large intestine | | $D_9$, $D_{10}$, $D_{11}$, $D_{12}$? | Dorsolumbar pain in cancer of the intestine or mesentery |
| Rectum | | $S_2$, $S_3$, $S_4$ | |
| Liver | | $D_8$, $D_9$, $D_{10}$? | |
| Gall-bladder | | $D_5$?, $D_6$, $D_7$, $D_8$, $D_9$, $D_{10}$? | In gall-stones pain is referred mainly in the 8th and 9th costal interspaces, less frequently in the 9th and 10th. |
| Kidney and renal pelvis | | $D_{10}$?, $D_{11}$, $D_{12}$ | Girdle pains in malignant disease of the kidney. |
| Ureter | | $D_{11}$, $D_{12}$, $L_1$ | Girdle pains and pain referred toward the nerves of the lumbar plexus in nephritic colic |
| Bladder | Muscular layer | $D_{11}$, $D_{12}$, $L_1$ | Dorsolumbar pain in cystitis. |
| | Mucous layer | $S_2$, $S_3$, $S_4$ | Pain the result of irritation by foreign bodies (stones, etc.) |
| Uterus | Body | $D_{10}$, $D_{11}$, $D_{12}$, $L_1$ | Dorsolumbar pain in parturient women. |
| | Cervix | $S_2$, $S_3$, $S_4$ | Pain due to inflammatory states and tumors of the cervix. |
| Testicle or ovary | | $D_{10}$ | Dorsolumbar pain in tumors or tuberculosis of the reproductive glands. Referred girdle-pain in cysts of the ovary |
| Epididymis<br>Fallopian tube | | $D_{11}$, $D_{12}$, $L_1$ | Dorsolumbar pain in orchitis, epididymitis, or suppurative salpingitis. |
| Prostate | | $D_{11}$, $D_{12}$, $L_1$ and $L_5$?, $S_1$?, $S_2$, $S_3$ | |
| Pleuræ and peritoneum | | | Referred pains extend along the course of the peripheral nerves, and are associated with deep-seated pain confined to the area actually involved. |

## PLETHORA. [πλήθειν, *to be full.*]

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The terms "plethora" and "plethoric," freely used in the clinical medicine of olden times, are not to be found in the standard treatises and text-books published in the course of the last forty years. In this fact lies one of the inevitable weaknesses of the prevailing nosology, which, soundly based as it was upon the pathologic conception of a certain clinical picture corresponding exactly to a certain definite organic lesion, found itself completely at a loss when required to classify correctly the functional symptom-complexes attendant upon morbid physiology. It was obliged to yield, unwillingly, in some instances, and attempted to associate a symptom-group with a definite lesion—not always, nor even frequently, succeeding in its endeavor One need merely recall the countless "lesional" theories of angor pectoris, such as the neuritis theory and the theories of coronary arteritis, of aortitis, of myocarditis, etc. As for certain other conceptions, such as the morbid temperaments, constitutional morbid predispositions, the "preorganic" stages of various diseases, and the "boundaries of the disease," according to Héricourt's very *justifiable expression* they were *deliberately* jettisoned from the nosological field as it was formerly accepted.

This opposition between that which the author has deliberately—and without overlooking the inaccuracy of the terms when taken in the strict sense—designated as functional nosology and the realm of lesional or organic nosology accounts in part for the frequently recorded lack of harmony between hospital practice and private practice. Hospital practice deals almost exclusively with lesional cases suffering either from acute disorders or from chronic, long-standing, lesional, inveterate, incurable disorders that have reached the stage of organic decompensation, such as advanced tuberculosis, arteriosclerosis, interstitial nephritis, cirrhosis of the liver, tumors, etc. Private prac-

tice, on the other hand, deals chiefly with functional cases suffering from morbid affections or tendencies of relatively recent advent and generally curable, such as pretuberculosis, hyposphyxia, plethora, transient or mild forms of cardiorenal insufficiency, active or passive congestion of the liver, etc. Hospital medicine, which hitherto has afforded the most clearcut material utilized in standard systems, deals mainly with extreme forms of disease, very often perfectly established and with highly definite outlines. Private practice generally supplies for the physician's observation incipient clinical types, an infinitely greater range of abnormal conditions, and morbid tendencies sometimes as yet barely outlined; yet any one can see that it is precisely upon the detection of these premonitory stages of presclerosis, of pretuberculosis, of cardiac, renal, or hepatic insufficiency, etc., latent or incipient, that the efficacy of our therapeutic endeavors depends.

A concise consideration of *plethora* will illustrate this assertion as a concrete clinical example

*Plethora* (πληθώρη, from πλήθειν, to be full) constitutes a very distinct and common clinical type In its simple, uncomplicated form, it strikes the eye by virtue of the subject's flourishing, often ruddy, supernormal, "overfilled," "plethoric" appearance:

The plethoric subject is, in truth, by no means a sick person in the ordinary sense of the term; on the contrary, apart from certain minor, intermittent ailments such as skin disturbances, hemorrhoids, etc., he enjoys a flourishing, seemingly perfect state of health; he even shows an unusual functional activity characteristic of more intense vitality; he is polyphagic and his digestive functions are admirably carried out (as, indeed, they are in diabetic, gouty, and obese subjects); he is polydipsic and polyuric (like the diabetic and gouty); his skin is ruddy and his general appearance robust; without his being actually obese, his weight is nevertheless above the normal (96 kilograms with a height of 187 centimeters, 74 kilograms to 166 centimeters, etc.); his powers of endurance are great; he is unusually active and the amount of work he does may be far above the average (as in many gouty and diabetic individuals).

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The terms "plethora" and "plethoric," freely used in the clinical medicine of olden times, are not to be found in the standard treatises and text-books published in the course of the last forty years In this fact lies one of the inevitable weaknesses of the prevailing nosology, which, soundly based as it was upon the pathologic conception of a certain clinical picture corresponding exactly to a certain definite organic lesion, found itself completely at a loss when required to classify correctly the functional symptom-complexes attendant upon morbid physiology. It was obliged to yield, unwillingly, in some instances, and attempted to associate a symptom-group with a definite lesion—not always, nor even frequently, succeeding in its endeavor. One need merely recall the countless "lesional" theories of angor pectoris, such as the neuritis theory and the theories of coronary arteritis, of aortitis, of myocarditis, etc. As for certain other conceptions, such as the morbid temperaments, constitutional morbid predispositions, the "preorganic" stages of various diseases, and the "boundaries of the disease," according to Héricourt's very justifiable expression they were deliberately jettisoned from the nosological field as it was formerly accepted

This opposition between that which the author has deliberately—and without overlooking the inaccuracy of the terms when taken in the strict sense—designated as functional nosology and the realm of lesional or organic nosology accounts in part for the frequently recorded lack of harmony between hospital practice and private practice Hospital practice deals almost exclusively with lesional cases suffering either from acute disorders or from chronic, long-standing, lesional, inveterate, incurable disorders that have reached the stage of organic decompensation, such as advanced tuberculosis, arteriosclerosis, interstitial nephritis, cirrhosis of the liver, tumors, etc. Private prac-

and elaborates When the plethoric subject has exhausted the cardio-arterio-renal reserve powers which have up to that time maintained and insured his abnormally high level of physiologic performance, he passes gradually into a *stage of angiospastic presclerosis*, during which he experiences intermittent attacks of renal insufficiency with retention, clinically expressed in paroxysmal high blood-pressure, hydremia, and their consequences—anginose pains, suffocative sensations, pseudoasthma, transient reduction of urinary output, etc. If this condition of presclerosis, which is still in large measure a reducible condition, *is not set right*, a definitive and practically irremediable *arteriorenal sclerosis* becomes established. The subject is no longer merely a patient, but a permanent invalid

The foregoing concise account will have set before the reader the prime importance of the syndrome, plethora, which, correctly interpreted and treated, will obviate in many instances an otherwise refractory condition of general tissue deterioration [1]

[1] For further details, see MARTINET *Pressions artérielles et viscosité sanguine*, Masson, 1912, *Clinique et thérapeutique circulatoires*, Masson, 1914, and in the present work, the section on *High blood-pressure*

In short, in the absence of any sort of illness on his part, one would almost be apt to state that the plethoric is a supernormal person or "superman" from the standpoint of body physiology. His more powerful, hypertrophied heart contracts more forcibly, leading to an unusually high systolic and differential blood-pressure His blood, more rich and less dilute, exhibits a higher viscosity and frequently an enhanced number of blood cells. His kidneys, adapted to more active circulation and nutrition, excrete unusually large amounts of water, salt, urea, uric acid, etc, and of urine, which often shows high acidity and high specific gravity. His digestive glands, copiously supplied with blood, produce unusually large amounts of secretion, causing polyphagia, polydipsia, polyuria, plethora, etc.

The plethoric subject is thus not, strictly speaking, an abnormal, but rather a supernormal individual, clinically characterized by his flourishing appearance, his supernormal body weight, and his high blood-pressure and blood viscosity.

He is predisposed, however, to obesity, to diabetes, to gout, and to urinary lithiasis, of which he already presents certain typical features as regards body conformation and functionation. He is predisposed to the development, sooner or later, of cardiovascular-renal fibrosis The chief advantage, indeed, of a diagnosis of true plethora founded on the symptomatic triad, overweight, high blood-pressure, and high viscosity (with their corollaries, high urinary acidity and specific gravity), is that it points, long before any recognized and ordinarily listed morbid manifestation has appeared, to the presence of an abnormal tendency which, at the time, is still susceptible of relatively easy correction before any irreparable organic change has become established (see *High blood-pressure*).

In over one-half of all cases of *plethora* duly substantiated by the presence of overweight and high blood-pressure and viscosity, the plethoric state will be found associated with a recognized metabolic disease, *viz.*, diabetes (see *Glycosuria*), obesity (*q v.*), gout (see *Joint pains*), or urinary lithiasis (see *Lumbar region, pain in*).

Plethora constitutes, furthermore, a premonitory stage in arteriorenal fibrosis or arteriosclerosis, which it precedes, heralds,

factory balance between the two is noted. Their urinary output per centimeter of differential or pulse pressure is, moreover, normal, *viz.*, 250 cubic centimeters (see *High blood-pressure*).

*Polyuria following injections of saline or glucose solutions, the administration of diuretic agents, or the institution of other diuretic measures* likewise presents an obvious exciting cause, but should stimulate the physician to record accurate and carefully made observations such as will ultimately permit of our formulating a practical and rational system of pharmacodynamics as related to the diuretic agents.

* * *

**Spontaneous, accidental, temporary polyuria** is met with particularly under two well-defined conditions

1. **After paroxysmal nervous attacks**, especially among hysteric subjects, epileptics, and exophthalmic goiter (post-hysteric, post-epileptic, and hyperthyroid polyuria), and even after ordinary spells of nervous excitement among naturally "nervous" subjects, *i e*, persons with unusual, excessive nervous reactions to stimuli

2 **In the critical stage of febrile diseases** and more particularly in the stage of resolution in pneumonia, influenza, bronchopneumonia, pleurisy, etc In these it constitutes, as a rule, a favorable prognostic sign of the greatest importance and which generally marks the change of trend of the disease from a fatal to a favorable termination

The foregoing types of polyuria are, as will have been noticed, of considerable practical importance, but those to follow are far more significant still

* * *

**Spontaneous, habitual, chronic, or at least recurring, polyuria.**—The most commonly encountered forms are those of renal fibrosis (interstitial nephritis), diabetes, and chronic diseases of the urinary tract The several different forms may be intermingled, but ordinarily they are completely dissociated and their recognition is easy, rapid, and elementary.

## POLYURIA.

[πολύς, *much*; οὖρον, *urine*.]

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The first thing to do in a patient who states that he passes much urine or in whose case there is reason to believe that polyuria exists is to *make sure that the condition is actually present*. Many patients confuse the terms frequent urination (pollakuria) and free urination (polyuria). As a matter of fact, no necessary and intimate relationship exists between these two manifestations. The practitioner should therefore have the patient carefully and systematically collect the twenty-four hour urine in one or more two-liter (or two-quart) containers Polyuria cannot be said to exist unless the twenty-four hour output (*e g.*, from 8 A.M. to the following 8 A M.) materially exceeds 1.5 liters, which is the normal average amount in an adult on a normal diet. The average degree of polyuria which is by far the most frequently met with is that ranging between 1,750 and 3 liters. In exceptional instances, such amounts as 4 to 6, 8, 10 liters, or even more, have been and are encountered.

* * *

Little space will be devoted to the subject of induced polyuria, whether of physiologic origin and due to spontaneous *ingestion of fluid in large amounts,* as in polydipsic subjects, or of therapeutic origin, in conformity with the physician's orders Where this form of polyuria exists care should be taken at least to ascertain approximately the total amounts of fluid ingested and of urine excreted Only by a comparison of these two amounts can a reliable conclusion be reached as to the eliminatory power of the kidneys as regards water (see *Functional examination of the kidneys: Induced diuresis*)

This applies to plethoric subjects; they eat much food and drink much fluid and consequently pass a large volume of urine, and if a careful comparison of their ingesta and excreta is made, a satis-

(*a*) In 9 cases out of 10 there is glycosuria, signifying *diabetes mellitus*, the cause of which is thereupon to be sought

(*b*) In 1 case out of 10 *glycosuria is absent*, but there is excess of nitrogen, of chlorides, of phosphates, etc In this event the condition is termed *diabetes insipidus*, and in this connection one is confronted with one of the more complex aspects of polyuria, since it borders on the condition known as *renal hyperpermeability* —a syndrome exactly opposite to the hypopermeability which results from renal fibrosis[1]—as well as on *amyloid degeneration of the kidney*, on *idiopathic* (or cryptogenic) *polyuria*, sometimes of hysteric origin (and what now remains of hysteria?), and on *polyuria symptomatic of nervous disorders*, especially bulbar, tumors of the medulla, disseminated sclerosis, general paralysis, hemorrhage, softening, tumors of the pituitary, etc Discussion of all these allied and varied conditions would lead us too far afield; let the mere mention of their possible clinical occurrence here suffice.

3 **Polyuria in "urinary" cases.**—In this group are included the instances of polyuria almost constantly met with in the course of the chronic diseases of the urinary tract, such as hypertrophied prostate, stone in the bladder, chronic cystitis, pyelonephritis (especially of calculous origin), etc

Its mode of production is undoubtedly complex, the symptom being the result of a reflex stimulation (possibly vasodilator), of secondary interstitial changes in the kidney with secondary high blood-pressure, and probably of other as yet poorly understood factors.

At all events, such a polyuria seldom exceeds 2 or 2½ liters. Only very exceptionally is the urine passed clear as in the preceding categories The associated infection of the urinary tract makes of it a *cloudy polyuria*, in which the turbidity is due to pus in the urine and sometimes, likewise, to alkalinity (or hypoacidity) and phosphaturia In relatively recent cases the urine clears up on standing, the pus and other solid substances forming a voluminous deposit at the bottom of the receptacle; in inveterate cases, on the other hand, with more advanced renal lesions, the urine remains cloudy, with a much less abundant sediment

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[1] See MARTINET· *"Clinique et thérapeutique circulatoires,"* section on *Renal hyperpermeability*

**1. Polyuria in renal fibrosis (interstitial nephritis).**—The patient is of ripe or advanced age. Usually he has a moderate degree of polyuria (1800 to 2000 cubic centimeters), *with pollakuria and nycturia* The urine passed is light colored, of low specific gravity (1014 or less), with small proportions of urea and chlorides, and traces of albumin, frequently so small as to preclude determination, or even entire absence of albumin

Sometimes, and even often, the polyuria is accompanied by the ordinary signs of arteriosclerosis: Cardiac hypertrophy, accentuation of the second sound at the base, or even gallop rhythm, together with sinuous or sometimes actually hardened peripheral arteries; at a more advanced stage. Hemorrhages in various situations, as in the retina, from the nose (epistaxis), etc.

There exists, furthermore, a sign which in the author's view is pathognomonic of renal fibrosis in the stage of eusystoly, *i.e., of adequate cardiac compensation. In this stage there is always* a high systolic pressure, a high pulse pressure, and also frequently a high diastolic pressure. Determination of the quotient $\frac{H}{p}$ of the actual twenty-four hour output of urine, H, over the pulse pressure, *p*, as estimated with the Pachon instrument in the sitting patient in the later morning hours or in the afternoon, yields in the normal subject a result equal or superior to 250 cubic centimeters. In the patient with interstitial nephritis the same calculation yields constantly and permanently a result more or less inferior to 250 cubic centimeters, and the lower the figure, the more pronounced the process of sclerosis. *By accident and in a* strictly transient manner, such a figure may be recorded in an angio-spastic case, but never lastingly and permanently

**2. *Polyuria in diabetics*** —*The patient is generally* in middle adult life and presents a flourishing appearance The polyuria is usually more marked than in interstitial nephritis, amounting to 2 liters or more. The urine is more highly colored, and at times even deeply tinted; it is always of high specific gravity, *viz*, 1020 or above, 1030, 1034, etc. *Whether glycosuria is present or absent, this feature alone is almost sufficient to differentiate the* **polyuria of low specific gravity of nephritis from the diabetic polyuria with high specific gravity.**

# PRECORDIAL PAIN.

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There are many patients who complain of pains in the region of the heart and believe, therefore, that their heart must be affected. As a matter of fact, precordial pain, while sometimes of cardiac origin, is far more frequently of extracardiac origin. The commonest among these extra-cardiac causes are dyspepsia, aërophagia, neuroses, and more particularly the so-called "psychosplanchnic neurosis;" in addition, a host of other factors may on occasion be operative.

The fact is that the precordial region is anatomically highly complex.

The precordial parietes comprise, from before backward:

1. *The skin, subcutaneous cellular tissue, mammary gland, and subjacent muscles, in particular the pectoralis major*

2. *The chest wall proper, consisting of muscle, bone, and cartilage, and including the sternum, ribs, costal cartilages, together with the costal interspaces and their vessels and nerves*

3. *The pericardium and heart*

4 *Anteriorly, the tongue-like projections of the pleuræ and lungs between the pericardium and the thoracic wall*

5. *Posteriorly, the heart is ensconced in its mediastinal recess in more or less direct relationship with the esophagus, the descending aorta, and the mediastinal lymph-glands*

6 *Above, it is prolonged by the great vessels at the base, viz, the aorta and the pulmonary vessels*

7. *Below, it rests on the thin diaphragm, which alone separates it from the fundus of the stomach.*

8 *Laterally, it is in relationship with the mediastinal pleura, the lungs, the phrenic nerves, and the vessels to the diaphragm.*

There is not one of these structures which may not be the source of pain in the precordial region, some commonly, like the stomach, others exceptionally, like the mammary glands

This cloudy form of polyuria may be met with in *renal tuberculosis*, especially when in an advanced stage. At the beginning a copious, clear polyuria is more frequently observed, with traces of albumin, phosphaturia, and quite frequently with slight, manifest or occult hematuria, the latter being examined for by centrifugation and examination of the sediment for red blood cells.

* * *

Such are, for practical purposes, the three main varieties of chronic, persistent, lasting polyuria. Yet, as in all other clinical groupings, there remains, in last analysis, a residue of cases still obscure and which lend themselves poorly to any satisfactory didactic description. This is the group alluded to above in connection with the polyuria of diabetes insipidus, *viz*, the *essential* or *idiopathic polyurias*, or better, the *cryptogenic* polyurias, the latter term being more in accord with our present state of knowledge, since we are ignorant of their cause and even of their probable mode of production. Most of the case reports refer to young subjects, twenty to forty years of age, frequently alcoholic, and nearly always hysteric The onset is almost abrupt, with frequent urination. The amounts reported by many of the authors are so amazing, *e g*, 10 liters, 15 liters, or even 30 liters, that they inevitably suggest the "Tales of Hoffmann" and that it is difficult to believe that there are not among them some instances of "colossal" faking. Personally, the author has never observed any amount approaching the above figures. The highest amount recorded has been 7 liters, already a remarkable figure, and even in this case the circumstances as regards supervision of the patient, although a rather close watch was kept, were not such as to exclude all possibility of faking on the part of the patient—an irresistible tendency in such subjects, who are always going after that which is excessive and extraordinary and seeking mainly, like the members of certain schools of art, to "astound the physician." "The spider," said Marcus Aurelius, "takes pride in catching a fly; another creature takes pride in catching a hare; another, in catching a sardine, another, in destroying a wild boar; another, in killing Sarmatians" Another, we may add, in . . .

Where will pride not seek its outlet!

This is the ordinary extra-systole or premature beat, a condition frequently encountered and recorded. It is rather a transitory discomfort than an actual pain. For its clinical interpretation the reader is referred to the section on *Arhythmia.*

2. Pain localized about the apex of the heart, recrudescent with each cardiac contraction, and with an exacerbation at the same point when a rather deep inspiration is taken This is the typical pain of pleurisy involving the precordial cul-de-sac, often readily detected by auscultation (localized rub during inspiration and expiration, ceasing during the periods of apnea)

The *pain* of *pericarditis sicca*, sometimes rather similar, is, as a rule, easily differentiated by the persistence of the friction sounds even during apnea and their synchronism with the heart beats

They may be accompanied by local tenderness

3 Pain more or less localized at the apex, with superficial darts and radiation toward the left lateral and posterior portion of the chest. This is the typical pain of intercostal neuralgia. The three characteristic points of tenderness should be examined for.

The various possible causes of the pain should be sought, *viz.*, rheumatic fever, Pott's disease, beginning meningomyelitis, osteoperiostitis, etc.

4. Pain as of a surface bruise or muscle cramp in the left side of the thorax, accompanied by tenderness on pressure or pinching of the muscles, accentuated by movements of the left arm, and allayed by rest of the extremity. This may be a myalgia of the pectoral muscles due to overuse of these muscles, traumatism, or exposure to cold

5 A sensation of intrathoracic distention, of a "large heart," of a too narrow chest, frequently accompanied by some degree of anginose discomfort and by dyspnea increasing upon exertion, walking, and climbing All grades may be encountered, from a slight, transient pain behind the sternum coming on upon marked exertion and ceasing upon termination of the latter, to a continued pain with persistent anginose discomfort and increasing dyspnea. In these cases the physician's attention should be definitely directed to the *myocardium*, the condition is probably a myocardialgia; the well-known dyspnea on exertion is present; only a concrete clinical

Clinically, a correct diagnosis can, as a rule, be quickly reached on the basis of three circumstances, *viz.*, *the nature of the pain*, *the time at which it appears*, and *the associated signs*.

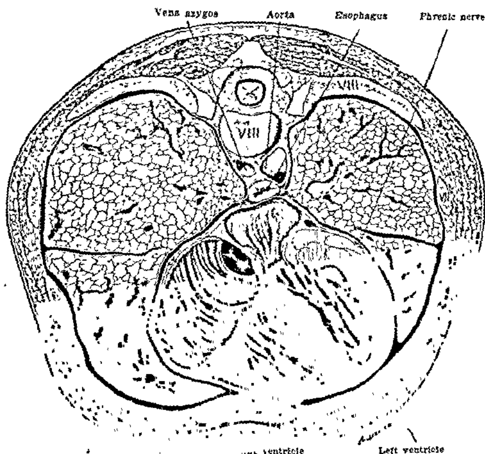



Fig ...zontal cross-section of the chest of a new-born infant through the eighth dorsal vertebra (*Poirier*)

### (a) NATURE OF THE PAIN.

This may, for practical convenience, be divided into the following six varieties:

1. A sensation as of a "misstep" or momentary arrest of the heart, frequently accompanied by a precordial thump with a slight feeling of constriction at the apex of the heart and of transient faintness, and sometimes preceded by a sensation of constriction in the esophagus

of a neurotic or aërophagic patient. Hence, he should carefully analyze the symptom before making, even merely in his own mind, a diagnosis of angina, and should avoid expressing any sort of a prognosis until after painstaking, repeated, and prolonged examination. The author has seen patients with extensive aortic lesions and subject to apparently alarming anginose attacks live ten, twelve, fifteen years or longer, in some instances even with complete intermissions of several years' duration.

The reader may have noted how difficult it has been to dissociate in a clear-cut manner the pains of the myocardialgic type from those of the type of angina pectoris Indeed, the relationship between the two is very close and there are insensible gradations. This has been well and forcefully expressed by Esmein as follows:

"There now appears the cardinal symptom of insufficiency of the left ventricle, *painful dyspnea.* This term, which certain methodical minds might be tempted to criticize, is the one most appropriate for designating the underlying functional disturbance existing in these subjects. From the beginning, the *dyspnea on exertion* (manifest upon climbing stairs, hilly streets, etc ) is accompanied by *painful sensations* behind the sternum and in the epigastrium, and these painful sensations are, from the start, of a subjectively alarming character, although ephemeral and rapidly allayed by rest.

"Then, there appears the *dyspnea of recumbency,* coming on abruptly on the approach of or during sleep, and likewise accompanied by *subjectively alarming* precordial *pains, frequently* radiating to the back, shoulders, and arms Sometimes the dyspneic factor distinctly predominates over the pain factor; there exists then an asthmatoid dyspnea or, employing the questionable term which is nevertheless consecrated by usage, a cardiac pseudoasthma.

"When the main features of these painful manifestations are reflected upon, a single word at once comes to mind, *viz., angina pectoris:* Shall we, on the basis of slight symptomatic variations relating to the duration and intensity of a symptom, perpetuate former errors and separate these anginose pains, this alleged *angina minor* (from which one is not expected to succumb!), from true angina pectoris, which causes death? The clinical course of the disturbance, here again, will bring us back to the truth. It is not rare, indeed, to

study and the use of appropriate tests will, however, permit of estimation of its degree, significance, and seriousness. It may just as well be the result of aerophagia, interfering by direct pressure with expansion of the myocardium, as the result of cardiac neurosis, bringing about an angiospastic attack with excess of work imposed upon the heart, or as the result of true myocardial degeneration leading to progressive cardiac insufficiency.

6 A sensation of violent constriction of the thorax, of squeezing of the chest as in a vice, with a constricting pain, clawing sensation, spasm, or strangling extending from the post-sternal area to the upper part of the chest, sometimes with radiation to the left arm on its ulnar aspect, more rarely along both arms or only on the right, paroxysmal, and accompanied by anginose distress of varying degree which may go so far as to yield a subjective impression as of imminent death. This is the typical pain of angina pectoris, which may be encountered in varying degrees. As in the preceding type, its significance, degree and seriousness can be determined only by accurate clinical analysis. Angina pectoris, or better the anginose syndrome, is, indeed, not only witnessed in all grades but may be brought on by the most varied causes, from the mildest kind of aerophagia to the most serious aortitis with coronary disease and myocarditis Hence the much criticized classification of olden times into pseudoangina pectoris which does not threaten life and true angina pectoris which kills the patient. This simple conception is incorrect in that such a clean-cut division is a practical impossibility and by no means harmonizes with clinically observed facts. What is true, however, is that it is the physician's duty to ascertain, by painstaking clinical analysis, that which underlies the anginose syndrome and, together with its intensity and its cause, its mildness or seriousness, to estimate its gravity in the individual case —an extremely variable quantity.

The diagnosis of angina pectoris is often made with disconcerting freedom One cannot sufficiently warn the practitioner of the twofold moral responsibility he undertakes either in overlooking the seriousness of an anginose syndrome which is the clinical expression of a lethal cardioaortic disease or in holding the terrible sword of Damocles of sudden death over the head

the same meaning as dyspnea on exertion. Much oftener, however, it is merely the symptomatic expression of a *neurotic dyspepsia* in which contact of food with a hyperesthetic mucous membrane reflexly brings on various cardiac disturbances, such as palpitation, painful tachycardia, premature beats, angor, dyspnea, and anginose attacks, or of *aërophagia* giving rise, through pressure on the heart through the diaphragm, to a mechanical hindrance to relaxation of the heart in diastole.

Emotional factors act similarly through angiospasm, which is one of their essential manifestations. The sensation as of a

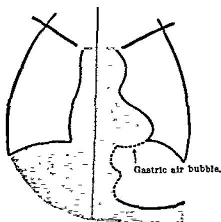



Fig. 851—Case 1279 H, 1900, 178 cm.; 80 5 kilogr Pulse rate, 92 Pressures, 140/90 Aërophagia Dyspnea on exertion Precordialgia.

"large heart" or of a "constricted heart" is one of the constant attributes of depressing emotions such as worry, apprehension, grief, and pain. Painful tachycardia, with the heart "palpitating" and "driven," is one of the constant attributes of abrupt, violent emotions such as surprise, emotional shock, fear, etc. The angina syndrome and mental anguish often form a reversible couple. This amounts to saying that painful emotional dyspnea is a common, physiologic occurrence devoid of any marked pathologic meaning Yet, upon analysis, it may reveal itself as meaning myocardial asthenia, in common with dyspnea on exertion, or as meaning a neurotic visceral pain It is only upon supplementary

find appearing, in subjects who had previously presented merely this rather attenuated syndrome, the major disturbances of insufficiency of the left ventricle: *Angina pectoris*, the most characteristic, and also *pulmonary edema*, a description of which need not here be given. Since the investigations of Merklen, it is no longer possible to overlook the bond which unites the painful dyspnea of high pressure cases, angina pectoris, and pulmonary edema, as well as the relationship of these symptom-groups to left ventricular insufficiency."

Yet, in the *anginose syndrome*, while it appears that one may ordinarily attribute the anxiety, dyspnea, and sensation of thoracic constriction to left ventricular insufficiency with dilatation or a tendency to dilatation, the clawing sensation beneath the sternum, the strangling, and the interscapular pain radiating to the trachea seem rather dependent upon traction on the nerve plexuses around the aorta. The anginose syndrome, as a rule, does actually result from a combination of these two factors, *viz.*, the aorticonervous factor and the myocardial factor.

### (*b*) TIME OF APPEARANCE OF THE PAIN.

The time relations of the pain, and the circumstances under which it occurs, sometimes supply extremely valuable diagnostic indications

Precordial pain accompanied by dyspnea, appearing only **upon exertion** (walking up inclines, carrying heavy weights, etc.) and disappearing upon rest, nearly always indicates an *at least relative insufficiency of the myocardium.* The same is frequently true of continuous dyspnea with a sensation of pressure in the chest, increased by exertion; this is the form regularly met with in the advanced stages of decompensation in *cardiopulmonary disorders*, such as emphysema, tuberculosis, chronic bronchitis, chronic endocarditis, cardiorenal fibrosis, etc. This long accepted conception of a "dyspnea on exertion" is of the greatest clinical service provided it is accurately observed, seen, and even measured (see *Functional heart tests*).

A hearty meal acts in the same way as marked exertion, and may lead to the appearance, in cases of cardiac insufficiency, of dyspnea, a sensation of pressure in the chest, subjective alarm, and even an attack of angina. Thus, **post-prandial dyspnea** may have

In practice the following varieties are more or less readily distinguished:

**Functional extra-systoles**, reflex (aërophagia, dyspepsia, or neurosis) or toxic (gout), intermittent temporary extra-systoles, unaccompanied by any general circulatory disturbance, are devoid of any significance as regards the heart and circulation. They diminish or even disappear upon exercise They are generally the symptomatic expression of a cardiac neurosis or dyspepsia, or of both. They constitute one of the most frequent attributes of the psychosplanchnic neurosis

**Organic extra-systoles**, as a rule practically permanent, are the outward expression of myocardial disease and are accompanied by the ordinary signs of myocardial and vascular degeneration already repeatedly enumerated (changes in blood pressure, stasis symptoms, dyspnea on exertion, evidences of aortic degeneration, orthostatic oliguria, nycturia, stasis edema, etc). They are increased or brought on by exercise and exertion. In this event the extra-systole is an evidence of myocardial degeneration which, taken in conjunction with other evidences, points toward the customary prognosis of myocarditis

**Dyspnea on exertion**, from the very fact of being so common, is of significance only by reason of the objective signs with which it is accompanied It may be and frequently is the subjective manifestation of an organic cardiopulmonary insufficiency due, *e g*, to an endocardial lesion (valvular disease), myocardial disturbance (fibrous degeneration), or pulmonary disorder (emphysema, chronic bronchitis, etc), it may, however, be merely the outward expression of aërophagia or of a neurosis

A careful search should therefore be made for:

1. The usual signs of valvular disorders, particularly mitroaortic (see *Circulatory procedures*).

2. The usual signs of pulmonary disorders, such as emphysema, chronic bronchitis, and lung congestion (see *Respiratory procedures*).

3 The usual signs of cardiac decompensation, *viz*, vesperal edema, latent edema at the bases of the lungs, orthostatic tachycardia, and orthostatic oliguria In this investigation one should, if need be, seek assistance from the circulatory functional test (see *Circulatory procedures*).

clinical analysis and by bringing together the coexisting morbid signs, however, that such a distinction can be made.

Finally, there remains the highly important subject of pains occurring mainly in the daytime or at night. This question is often rather hard to interpret. While, in its earlier stages, the painful dyspnea of incipient cardiac insufficiency is relieved by rest and recumbency, at a later period one may, on the contrary, observe the appearance, especially in cardiorenal cases, of a decubital dyspnea coming on abruptly at the approach of or during sleep and sometimes accompanied by subjective alarm and asthmatoid attacks

Again, these nocturnal manifestations, insomnia, subjective alarm, dyspnea, or even angina and cardiac pseudoasthma are ordinarily far more frequent, striking and dramatic in neurotics (cardiac neuroses) than in organic heart cases. Such psycho-somatic nocturnal disturbances are especially characteristic in neurocardiac patients (see below).

Consequently, this analysis of the subjective aspects of precordial pain and its several modalities, however valuable it may be and however systematically it should be carried out, must, from the standpoints of diagnosis, prognosis, and treatment, give place to the concrete, objective examination of the case, and to the search for coexisting signs, observation and correlation of which can *alone* permit of a well-founded and firm conclusion

## (*c*) ASSOCIATED SIGNS ALONG WITH PRECORDIAL PAIN.

The three least distressing varieties of precordial pain, myalgic, neuralgic, and pleuropericardial, having been rapidly, and as a rule very readily, excluded, there remain the three standard forms previously described: 1 **Extra-systoles.** 2 **Dyspnea on exertion.** 3 **Anginose syndrome.** Taken singly these conditions sometimes, in fact frequently, offer marked difficulties as regards a causal diagnosis

The associated signs alone will permit of making such a diagnosis, the symptoms themselves possessing only a very restricted meaning.

**Extra-systoles** are of no definite diagnostic significance; all depends upon the circulatory symptoms accompanying them.

4. **Angina pectoris in neuroses and psychoneuroses, eminently** benign as regards the heart, but of such practical importance that it seems advisable to devote considerable space in this section to its discussion and to reproduce *in extenso* an article on the cardiac neuroses published by the author some years ago (*Presse méd.*, Nov. 4, 1915).

## (*d*) THE CARDIAC NEUROSES.

Special significance was lent to the question of the cardiac neuroses by the late war More than two-thirds of the hospital cases labelled "heart disorder" belonged in this category.

And first of all, what is meant by the term cardiac neurosis? The practical, clinical definition of the condition seems simple: Cardiac neurotics are those subjects who, in the absence of any acute or chronic organic lesion of the heart or its coverings (endocarditis, pericarditis, or myocarditis), in the absence even of any true myocardial weakness, *e g*, congenital or constitutional weakness, or of any recognized lesion of the nervous system, suffer from a symptom-complex involving mainly the heart This definition excludes all the organic disorders of the heart, all cardiac manifestations dependent upon some *organic* nervous disorder, central or peripheral, and all temporary and evanescent cardiac manifestations of reflex origin and extra-systoles, *e g*, of digestive origin

With the ground thus cleared by a relatively easy process of clinical elimination, there yet remains an extensive, rather homogeneous group of cases—although further study of the cause may easily resolve it into sub-groups very variable in their pathogenesis and clinical course (cardiac neurasthenia, Graves's disease, etc). All these subjects have in common the fact of suffering from severe, manifold, and refractory manifestations in the cardiac region, and more generally, of disturbances of a circulatory nature, while nevertheless presenting upon examination an apparently complete integrity of the system referred to

As a matter of fact, it is these self-same *cardiac neuroses* which are accompanied by the most numerous and distressing *cardiac* or pseudocardiac *symptoms*: Squeezing or constriction of the heart, a distressful sensation of beating arteries or of "missteps" of the heart, an anginose feeling with pains radiating in the arm and neck

As in the case of the *extra-systoles, this systematic* and necessary investigation will lead to the differentiation of:

*Functional dyspnea on exertion, in the absence of organic disease of the heart or lungs* (as in aerophagia, dyspepsia, and neurosis), the result of mechanical pressure on the heart (gastric air bubble), of *reflex excitation* (*dyspepsia*), or of an erethistic psychosplanchnic predisposition (psychosplanchnic neurosis)

*Organic dyspnea on exertion*, due to cardiopulmonary insufficiency, the result, in turn, of an *appreciable, tangible lesion in the cardiopulmonary system*, associated with the hyposphyxic syndrome (see *Low blood-pressure*)

The previously emphasized relationship of dyspnea on exertion and the **anginose syndrome** suggests *a priori* that the same considerations shall apply to angor, and at bottom this is in all likelihood what the older authors understood without actually expressing it *in their much-criticized division into the pseudoanginas and the* true anginas. The author will take good care not to enter into any doctrinal discussion in this purely practical work.

The following exclusively clinical classification seems advantageous.

1. **Angina pectoris dependent upon an aortic lesion** (aortic aneurysm, aortic valvular disease, interstitial or specific aortitis, arteriosclerotic degeneration, or cardiorenal sclerosis), very frequently combined with chronic degeneration of the myocardium, yields a first rather homogeneous group of cases—cases of serious angina which may prove fatal, and always necessitating a very guarded prognosis, although the author has witnessed survival for ten, fifteen, or more years, even in the presence of very advanced aortic lesions (see *High blood-pressure*).

2. **Angina pectoris occurring in plethora, presclerosis, angiospasm, nicotinism, or gout,** generally much less serious than the preceding type, and very often curable provided the patient will submit to appropriate hygienic regulation (see *Plethora, High blood-pressure,* etc).

3. **Angina pectoris in aerophagia,** well described by Robin and Fiessinger, and actually rather often encountered by the author. While occurring *alone, such angina rarely assumes the* form of true major angina.

and lengthening of both ventricular diastole and systole; when very marked it is capable of inducing a pronounced slowing of intracardiac conduction or even auriculoventricular dissociation through inhibition of the bundle of His, as in the bradycardia and dissociation produced by digitalis Inhibition of the centers referred to leads to opposite results—a fact which by no means tends to facilitate, in clinical studies, inquiries as to that portion of the effects relating to each one of these nerves.

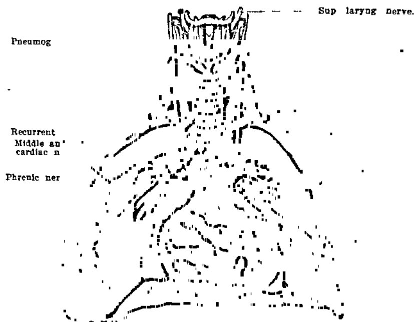



Fig 852—The nerves of the heart (*Hirschfeld*)

It should be added that the actions of both nerves extend to the peripheral circulatory structures, giving rise, as the case may be, to vasoconstrictor phenomena (or even angiospasm) or to vasodilator phenomena (or even vagoparesis), reacting, in turn, upon the heart either in a direct mechanical manner or indirectly and reflexly

The medullary centers, on the one hand, and the myeloganglionic nerve paths, on the other, are manifestly influenced alike by stimuli of psychic and special sensory origin (special sensory stimuli and concepts, images, recollections, emotions, etc.)

(nervous angor, dyspnea, constriction of the esophagus and neck, choking sensations, etc.), as well as by *equally numerous,* if not alarming, *objective signs,* such as *tachycardia, tachy-arhythmia,* extra-systoles, marked vasomotor instability (*involving both the* pulse frequency and the blood-pressure), fainting spells, profuse sweats, frequently cardiac hypertrophy, with a special vibrant quality of the first sound, at times even an intermittent systolic apical murmur transmitted toward the axilla and the left sternal border, and frequently accentuation, sometimes reduplication, of the second pulmonic sound, etc.

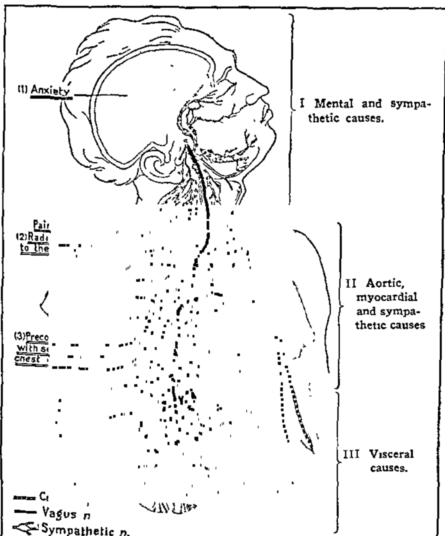
Finally, these conditions are ordinarily combined with unquestionable *neuropathic manifestations,* such as insomnia, nervousness, exaggeration of emotive reactions and of the reflexes, cenesthesic instability, headache or actual migraine, asthenia and abnormal excitability (irritable weakness of the nervous system, etc.), and sometimes manifestations of asthenic nervous overexcitability of other systems, as evidenced, *e.g.,* by gastrointestinal dyspepsia, asthmatoid phenomena, dermographia, and paroxysmal sweating.

This clinical picture, which is of exceedingly frequent occurrence, whether existing merely in faint outline or with its salient features pushed to their ultimate conclusion as in exophthalmic goiter, betokens and outwardly manifests better than could the most perfect experiment in physiology the intimate relationship existing between the nervous and circulatory systems. In the present state of our knowledge, this neurocirculatory interdependence may be outlined as follows:

Cardiovascular activity is controlled and regulated as a whole by the so-called vegetative nervous system, which consists, as is well known, of the vagus and the sympathetic with their bulbar centers. The vagus and the sympathetic are in a large measure mutually antagonistic. Stimulation of the sympathetic causes acceleration of the pulse (tachycardia), elevation of the systolic blood-pressure, and shortening of cardiac systole; when very marked it is capable of inducing an excessive, or even extra-systolic, pulse acceleration of the type of paroxysmal tachycardia. Stimulation of the vagus causes, on the other hand, slowing of the pulse (bradycardia), reduction of the systolic blood-pressure,

# ANGINA PECTORIS.

## SYMPTOMATOLOGY AND PATHOGENESIS




## PATHOGENESIS.

| I. | II | III |
|---|---|---|
| Mental causes: Anxiety, emotions, anxiety neurosis<br>Sympathetic causes: Sympathetic neurosis<br>Angiospasm { traumatic, organic, emotional | Aortic and periaortic causes: specific, gouty, ordinary<br>Coronary causes: Spasm See Angiospasm.<br>*Myocardial causes:* Sudden exhaustion of reserve power | Gastric causes: Aerophagia.<br>Visceral causes in general. |

and by cenesthesic stimuli arising through visceral sensation, which may be pleasant or unpleasant, or even painful.

The result of all this is an exceedingly close interdependence of the nervous and circulatory systems, which leads the circulation to react with extreme sensitiveness to any nervous stimulus, whether latent or manifest, conscious or unconscious. Clearly, the circulation reacts, perhaps more than any other system, to such incessant nervous traumatism as characterized the late war. How does it react under such conditions? Actually, in very diverse fashion, according to the individual.

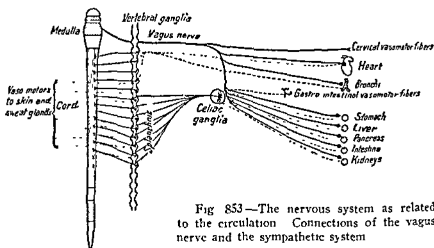



Fig 853—The nervous system as related to the circulation Connections of the vagus nerve and the sympathetic system

In most ostensibly normal persons the neurocardiac, or better, neurocirculatory reaction does not extend beyond a condition of temporary insomnia with accelerated pulse rate, nervous overexcitability with exaggerated reflexes, transient subjective alarm and tremor, and a few evanescent vasomotor and secretory manifestations, such as pallor or hot flushes, "goose flesh," sweats, temporary diarrhea, etc. Within a few days a more or less complete tolerance becomes established, the vegetative nervous system adapts itself to the new conditions, and the cardiac and vasomotor reactions are reduced to a physiologic minimum.

In others the emotional shock persists, leading to a protracted or permanent loss of neurocirculatory balance characterized by appearance of the symptoms already mentioned as constituting the cardiac neurosis.

As in the case of the reserve power of the heart-muscle itself, so the reserve power of the nervous system in these cases, including the capacity of resisting without collapse or even adapting itself to certain psychophysiologic stresses, varies exceedingly in different individuals Some nervous systems resist and adapt themselves even to the most severe trials, as in the case of the peasant who was buried for twenty-five days among the débris in a landslide and whose first words when finally set free were "Have the animals been saved?" In other instances the mere fact of entering the barracks is enough to upset the nervous system completely.

As a matter of fact, the author saw just as many, if not more cases of cardiac neurosis among subjects on duty far from the scenes of military action than among those who had actually been subjected to the gruelling life at the front

Huchard used to remark that "the physical heart is lined with a mental heart." The author some years ago read in an Italian periodical[1] the following naive yet truthful statement concerning subjects suffering from organic heart disorders and fully conscious of their infirmity: "Sustained by small doses of digitalis and *more particularly by their valorous spirits,* they were able to perform long and fatiguing missions, even as aviators"

This statement serves as a good paraphrase of Turenne's sublime remark to his own "beast" or body "You are trembling, carcass; you would be trembling much more if you knew where I am going to take you" A penetrating statement this was from the standpoint of body physiology We are powerless to restrain the reflexes of our medullary and spinal vegetative system, but an "energetic, valorous spirit" can always make its "beast" of a body advance, even if it is "shivering" and "palpitating" at the time.

Cases even occur in which the stimulus afforded by constant danger exerts a favorable effect on a preëxisting cardiac neurosis. Such is the personal case of Longhi, the Italian translator of Stokes's[2] classic work, which he describes as follows in his translation:

[1] MENDES *Manuale di medicina chirurgia di guerra,* Rome, 1915

[2] STOKES: *Malattie del cuore e dell' aorta.* Prima traduzione italiana del dott. A Longhi, Turin, 1858, p 223

## ANGINA PECTORIS (PATHOGENESIS).

**NEURO-VASCULAR COMPONENT** (Angiospasm).

Symbiosis { nervous *Sympathetic overactivity, predominantly in cervical region.*
vascular. *Aortico-coronary spasm.*

Causes:

FUNCTIONAL.—Emotive: *Anxiety the psychic primum movens.*
Reflex *Visceral, dental, gastric, etc. (in the predisposed)*
Toxic: *Tobacco, adrenalin*
Auto-toxic: *Diathetic, gouty.*

ORGANIC —Aortitis *Coronaritis* (?)
Periaortitis *Mediastinitis.*
Neuritis of sympathetic
Cervico-dorsal osteoarthritis.

---

**MYOCARDIAL COMPONENT** { Distention of left ventricle.
Exhaustion of reserve power.

Causes { Degenerative myocarditis *in general.*
Extracardiac mechanical causes (*aerophagia*)

Prognosis:

If the *reserve power of the heart is good Attacks not dangerous*
If the *reserve power of the heart is poor· Attacks serious or fatal* { Acute edema
Syncope

had entirely forgotten his cardiac discomforts and experienced merely slight dyspnea on running.

Such a turn of affairs is not exceptional, although, as a matter of fact, much less common than the converse sequence of events.

Considering only the heart symptoms, the *diagnosis* between cardiac neurosis and organic disease is not always easy. Auscultation may be puzzling and misleading, and various forms of arhythmia (extrasystoles, sinus arhythmia, etc.) may be observed in either instance; the same consideration applies to the customary hypertrophy of the left ventricle and even more strikingly to the subjective manifestations, *viz.*, dyspnea on exertion, sensation of constriction or actual angina, palpitation, phrenocardia, etc. Yet there are a number of differential signs.

(*a*) The first and most important, perhaps, is the *neurotic substrate* upon which the cardiac neurosis always develops. The cardiac symptom-group above referred to merely forms part of an always more or less pronounced neuropathic clinical picture, which is confirmed, in turn, by the family history and by extracardiac neuropathic manifestations, digestive and mental in particular.

(*b*) The second is the *relative frequency and importance of nocturnal symptoms,* such as insomnia, subjective alarm, dyspnea, and even anginose discomfort and cardiac pseudoasthma, which are far more common and striking and generally more dramatic than in subjects with organic heart disease. These characteristic and interesting nocturnal psychosomatic disturbances among neurocardiacs are in themselves deserving of a thorough study.

(*c*) The *neurocardiovascular instability and lability* constituting an outward expression of abnormal emotivity. The pulse frequency and blood-pressure exhibit surprising variations from the slightest disturbing causes. This is often true likewise even of the auscultatory signs which are far from presenting the relatively high degree of permanency and constancy of those of organic lesions. The changeableness as regards arhythmias is perhaps even more characteristic (Fig. 854).

(*d*) There may frequently be noted an *absence of the usual etiologic factors of organic heart conditions,* such as rheumatic,

"In the course of the winter of 1848, I was constantly troubled with palpitations and with the dejection of spirits which nearly always occurs in heart disorders. Tired of suffering, I went to see a *distinguished colleague*, a specialist in chest disorders, who told me that I had a hypertrophied heart—not a very serious condition in itself, but one which is *incurable like any other* dependent upon an organic lesion. He prescribed for me a line of treatment calculated to moderate the heart action and advised me not to give way to melancholia, as the disorder was not serious and would allow me to continue living a long time, even though I might experience slight discomforts from it

"On the very next day after my visit the revolution broke out in Milan, and in it I took a rather active part At the first gunshots that I saw and heard, my heart began to beat so strongly that I almost fell to the ground and feared lest I might be obliged to retire from the fight, not through cowardice, but *from physical weakness* *Shortly after, my heart became more* quiet and I found myself drawn into a skirmish in the course of which I had no opportunity to think of it. Subsequent to that day I led a very active life at the camp, at first as a volunteer and later as a Piedmontese officer of the bersaglieri, without ever being conscious of my heart action During the last nine years I have been in excellent health and have had no precordial pain I am convinced that in 1848 my discomforts were due principally to a temporary engorgement of the heart resulting from the sedentary life I led at that time, *spending eight or ten* hours at my desk each day, whereas in my youth I had been *accustomed to a very active life*"

The author knows of many neuro-cardiac subjects who no longer "felt their hearts" after the mobilization, as exemplified by an artillery officer aged forty-eight who, afflicted with palpitation, precordial pain, and angor, had been living since 1908 obsessed with the fear of aneurysm or angina pectoris and had, to the author's own knowledge, consulted about ten physicians in Paris, *none of whom had found anything more than* neurocardiac erethism and a moderate degree of hypertrophy. The author saw him again after he had been one year at the front, including three months in the trenches with the infantry, at which time he

tolic blood-pressure occurring when the subject rises from recumbency to the standing posture as well as after a series of carefully standardized exercises (20 dips with flexion of the lower extremi-

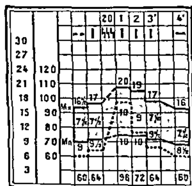



Fig 856—Normal subject as regards the circulation
H., 1875; 169 cm, 73 kilogr.

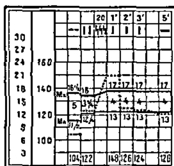



Fig. 857—Heart weakness.
H., 1893, 165 cm, 60 kilogr.

ties), demonstrates clearly the exaggerated vasomotor reactions (reflex overexcitability) in well-marked cases, as well as the con-

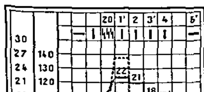



Fig 858.—Cardiac neurosis
H., 1878, 169 cm, 67 kilogr

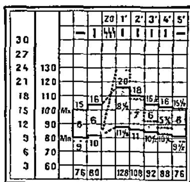



Fig 859—Cardiac neurosis
H, 1895, 169 cm, 64¾ kilogr.

siderable margin of reserve myocardial power usually present, as exemplified in the five annexed illustrations, two of which are from normal subjects, one from a case of weak heart, and two from cardiac neurotics (Figs. 855-859).

typhoid, syphilitic, diphtheritic, and other infections; plethora and autointoxication, gout, uricemia, etc.

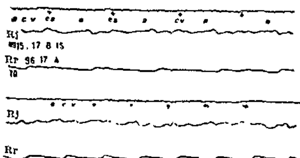



Fig 854.—Cardiac neurosis H, 1885, 174 cm; 73.6 kilogr, pulse, 96, pressures, 170/100; viscosity, 4

The two above tracings were taken about 1½ minutes apart. The first shows premature contractions imparting to the pulse tracing a bigeminal aspect In the second, all premature contractions have disappeared

(*e*) *High blood-pressure, systolic as well as diastolic,* is noted in the great majority of cases, in spite of the widely held view to the

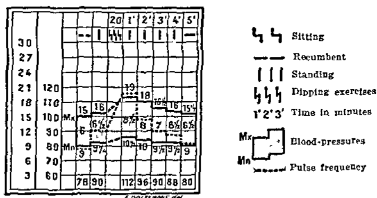



Fig 855—Normal individual H, 1893, 173 cm, 70 kilogr (*Mx* = systolic pressure, *Mn* = diastolic pressure; pressures given in centimeters of mercury)

contrary. There do occur, however, a few cases of cardiac neurosis with low blood-pressure; these are of two varieties, as the author proposes to show elsewhere

(*f*) Lastly, *the functional test of the circulation,* which consists in recording the changes in pulse frequency and systolic and dias-

1. One should make a careful analysis of the characteristics and *nature of the precordial pain* present. This initial procedure will orient the investigation *a priori* in some particular direction, but however searching the analysis may have been, *it will never lead to* a sound diagnosis, which can only be established by objective examination of the patient.

2. *Only a complete objective examination* (carried out as described in the section on *Systematized clinical examination*) *will permit of obtaining a more or less prompt solution of the diagnostic puzzle presented.*

In concluding this section it seems advisable to go over the subject of precordial pain summarily from a different aspect, *viz.*, by starting with the clinical conditions themselves and correlating with them the various forms of pain

## DISORDERS OF THE HEART AND AORTA.

**1. Disorders of the Cardiac Orifices. Endocarditis.**—(*a*) **During the stage of adequate compensation,** endocarditis can hardly be said to cause pain The frequency with which patients are unaware of their condition apart from periods of lost compensation is well known Yet the possibility must be recognized that among some nervous and hyperesthetic subjects there may eventually occur extra-systoles and a sensation as of cardiac distention, precordial hyperesthesia, or chronic precordialgia. Mackenzie doubtless had these cases in mind when he wrote: "Many subjects suffering from an actual heart lesion, such as may involve the mitral or aortic valve, exhibit evidences of exaggerated precordial sensitiveness This sort of thing is witnessed more particularly in women . . Attacks of very severe pain in the chest may be experienced . . . More frequently there is an unpleasant dull sensation . . . . The hyperalgesia may extend over a very large area and is sometimes very marked. . . . The pain may not be as distressing as in the more severe instances of angina pectoris, but it persists a longer time. . . . It is often associated with extreme tenderness of the tissues of the neck or left side of the chest, especially the left breast. Where the condition of tenderness of the skin and

In short, the *true, organic heart case*, when well compensated, reacts like a normal individual; when poorly compensated, he reacts like a case of heart weakness and yields a typical curve denoting cardiac insufficiency, *viz.*, a weak blood-pressure reaction, or even a reversed reaction, with slow return to the original state of equilibrium

The *cardiac neurotic* reacts to an exaggerated extent both as regards pulse rate and rise of blood-pressure, thereby affording an outward manifestation of his reflex cardiac and vasomotor over-excitability; on the other hand, his blood-pressure test shows no tendency toward myocardial insufficiency.

To be sure, an *organic heart case* may also be neurotic, and a *true neurotic* may be found suffering from cardiac debility, plethora, or even organic heart disease In such cases the functional test alone is frequently sufficient to demonstrate the simultaneous presence of the two disorders; when used in conjunction with the other methods of clinical investigation it nearly always enables the observer to distinguish that which attaches to the nervous system from that which is referable to the circulation, and thus to render a well-founded prognosis and institute a rational line of treatment based on a reliable physiopathologic conception of the case.

As far as the patient's availability for military service is concerned, it is not, as will readily be seen, the reserve power of the heart which is the criterion in the matter, ample reserve power being, as a rule, available It is rather the power of nervous resistance and reaction that should be investigated by appropriate methods.

The cardiovascular symptom-complex here constitutes but a single outward manifestation, albeit a highly important one, of an abnormal psychoneurotic state which dominates and governs the entire symptomatology as well as the prognosis.

* * *

From the foregoing description the reader will, it is hoped, have been led to realize both the complexity and the relative simplicity of the diagnostic problem which arises in connection with the precordial pains. Its solution may, on the whole, be concretely stated as follows:

left arm, or toward the right axilla and arm, or toward both arms, in accordance with the location and size of the aortic deformation This pain appears to be mainly of *periaortic, neurovascular origin*, being related to an inflammatory condition and distention of the nerve plexuses around the aorta

(*b*) **Pain as of constriction of the chest**, with a squeezing sensation, subjective alarm, a sensation as of impending death, the whole constituting the essential factor in the syndrome known as angina pectoris, while the preceding type, which so often accom-

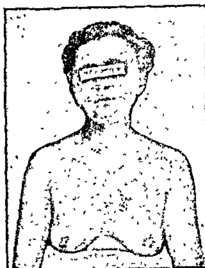


Fig 860—Aortic aneurysm resulting in an elevated pulsating tumor on the left side

panies it, is merely an auxiliary factor. This pain seems to be dependent upon deficient functioning of the heart muscle, or more specifically, an *acute insufficiency of the left ventricle*, which is painfully striving to overcome a resistance so marked as to exhaust its reserve power.

Dyspnea on exertion necessarily accompanies this type of pain The author has noted its absence, however, with the preceding type, in particular in a case of aneurysm of the ascending aorta which had eaten away the sternum, formed a pulsating tumor of the size of a hen's egg, and induced pain on the right side of the chest radiating to the right axilla and arm.

muscles has been tested by pinching them between the thumb and forefinger, the part remains sensitive for several hours"

As a matter of fact, this type of chronic precordialgia has seemed to the author *quite exceptional among well compensated and non-neurotic cases of endocarditis.* It is the rule, on the other hand, in the *cardiac neuroses* (see above), *hyposphyxia* (see *Low blood-pressure*), and *cardiac insufficiency* of whatever cause In the latter type of case, the mechanical factor, *distention of the heart,* seems to be more particularly concerned in its causation.

(*b*) **During the stage of lost compensation,** *ranging from a* mere reduction in the reserve power of the heart to actual heart failure, the picture witnessed is mainly that of the dyspneic *syndrome, from dyspnea on exertion to continuous dyspnea with* more or less pronounced anginoid phenomena and with the usual signs of impaired heart action, such as orthostatic oliguria, *cyanosis, edema, etc.*

2. **Myocarditis.**—Here the signs of lost compensation and cardiac insufficiency already enumerated constitute the main feature, sometimes with extra-systoles and attacks of angina

3 **Pericarditis.**—Pericarditis may either be completely latent or be accompanied by pain so slight and evanescent that some patients pay no attention to it. In the majority of cases pericarditis, especially when of the "dry" variety, induces precordial pain which is localized in the region about the apex or the sternum and is sometimes recurrent with the successive heart-beats Examination may demonstrate the presence of tender points the result of radiation along the phrenic nerve (*Guéneau de Mussy's points*), *viz*, the *lower point,* between the ensiform appendix and the border of the costal cartilages on the left side; the *intermediate points,* at the anterointernal portion *of the left costal interspaces, along the* sternal border, and the *upper point,* between the sternal and clavicular heads of the sternocleidomastoid muscle

4. **Aortitis.—Dilated Aorta.—Aneurysm.**—This disorder may be accompanied by three sorts of pain:

(*a*) **Pain behind the sternum and in the chest,** with or without radiation, either, as is most often the case, toward the axilla and

and angina in all respects similar to those already described. Again it should be emphasized that it is only through the anamnesis and especially by systematic and thorough clinical examination that these subjective disturbances can be traced to their actual causes.

7. **Cardiac Neuroses.**—This subject has already been referred to at such length that it seems unnecessary to discuss it again here. Any form of disturbance may be witnessed, from dyspnea on exertion to the anginal attack and from extra-systoles to permanent paroxysmal dyspnea

## DISORDERS OF STRUCTURES ADJACENT TO THE HEART.

1. **Pleurisy of the Precordial Diverticulum of the Pleura.**—In this condition there is pain localized at the cardiac apex, recurring with each heart beat and increased by rather deep breathing.

In nervous subjects it may lead to the production of reflex extra-systoles

2. **Aërophagia and Dyspepsia with Gastric Distention**—This is one of the commonest and yet one of the most frequently overlooked causes of precordial pain, the individuals concerned often being neurotic.

Dyspnea on exertion, most marked after meals, feelings of painful distention in the vicinity of the cardiac apex, extra-systoles, and attacks of pseudo-angina (angina pectoris of aërophagics), may be observed The time of occurrence of the pain (after meals), the tympany and increased size of Traube's space, actual observation of the aerophagia, and the absence of objective heart signs easily lead to the correct diagnosis if they are merely thought of

The orthoradioscopic view shown in Fig. 851 gives a good idea of the extent to which the heart can be pushed aside by the air content of the stomach

3 **Gaseous Distention of the Colon.**—The rather frequently encountered accumulation of gas in the splenic flexure brings about the same symptoms as have already been enumerated, and through the same process of mechanical displacement of the

(*c*) In the *stage of lost compensation* are added *permanent paroxysmal dyspnea* and the usual signs of heart failure.

5. **Arteriosclerosis. Disorders Attended with High Blood-Pressure.**—The forms of pain experienced in these cases are very similar to those referred to in connection with diseases of the aorta, which, indeed, is itself often involved in the process. Thus, there occur:



Fig 861.—Aortic aneurysm with precordial pulsating tumor.

(*a*) Pain of the type of dyspnea on exertion, which is the first to appear.

(*b*) Pain of the type of permanent paroxysmal precordialgia with dyspnea, subjective alarm, etc., of myocardial origin.

(*c*) Clawing pains behind the sternum, radiating to the periaortic region.

Interstitial nephritis and uremia are frequently accompanied by similar manifestations.

6 **Vasomotor Angiospastic Attacks.**—The attacks of high blood-pressure from vasoconstriction induced in some subjects by overwork, emotional impressions, or abuse of tobacco may eventually be accompanied by manifestations of precordial pain

# SLEEP, MORBID.

> *To die, to sleep;*
> *To sleep: perchance to dream*
> (SHAKESPEARE, *Hamlet*,
> Act III, Scene I)

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The crude analogy existing between sleep and death is obvious. It would lend itself most readily to a fascinating metaphysico-poetic discourse, most concisely and suggestively expressed in the Shakespearean epigram at the head of this section Such a discourse would, however, far transcend the definite purposes underlying the present work, and we shall have to preserve due limitations in this connection

* * *

Sleep may occur in very variable degrees and modalities. It may be divided, according to its intensity, into *somnolence, light sleep, profound sleep,* and *coma,* the latter, on the whole, being different from profound sleep only in a more complete loss of sensibility, in the impossibility of rousing the subject by artificial stimulation, and frequently in paralysis of the sphincters The differential diagnosis of the several forms of coma has already been dealt with in a special section (see *Coma*)

*To base conclusions on the degree of sleep present is not of great clinical value,* for in general one sees no definite lines of demarcation between the different grades of sleep and the same morbid cause—*e g*, toxic—may, according to the individual and the extent of the morbid influence present, yield any degree of hypersomnia, from simple somnolence to the most profound coma This is true, for example, in the case of alcohol, opium, and azotemia.

It seems more in keeping with clinical conditions and even cursory observation to base our *interpretation of the hypersomnias upon their clinical features,* and to divide them as follows:

*Hypersomnia* presenting the appearances of normal sleep and differing from it only in its time relations, duration, or depth.

diaphragm. The patients concerned suffer from spastic constipation; percussion elicits exaggerated resonance over the colon, and fluoroscopy sometimes yields an actual view of the gaseous accumulations. Liquid petrolatum and extract of belladonna are, as a rule, both diagnostically and therapeutically successful in these cases.

4. **Mastodynia,** *when localized in the left breast, may suggest,* albeit only approximately, precordial pain Any possibility of a mistake is readily obviated by the most superficial examination, including palpation of the breast and the observation of diffuse induration or of small scattered nodules, in conjunction with the absence of all true cardiac manifestations

5. **Intercostal neuralgia** is usually distinguished by virtue of the three characteristic points of Valleix: *Anterior,* by the side of the sternum; *lateral,* in the axillary line, and posterior or *paravertebral.* The neuralgia, moreover, would hardly tend to mislead unless located exactly in the precordial region It should be borne in mind that the term intercostal neuralgia, purely an anatomic expression, implies nothing as to the cause, which should be sought and may be either *toxic,* as in lead poisoning, gout, etc ; *infectious,* as in rheumatism, typhoid fever, etc ; *osseous,* as in osteoperiostitis, Pott's disease, etc ; *pleuropulmonary,* as in pleurisy, pneumonia, etc., or even *cardiomediastinal,* as in aortic aneurysm, hypertrophy of the heart, mediastinal tumor, etc One should never hesitate to resort to fluoroscopy in obscure cases.

6 As an exceptional cause, mention should be made of **tabetic pains.** Tabes dorsalis is frequently associated, as is well known, with more or less pronounced pathologic changes in the aorta, and some tabetics, quite naturally, may experience attacks of angina pectoris definitely of aortic origin In at least two tabetic cases, however, the author has observed paroxysmal and transient attacks of precordial pain which, since they occurred in alternation with other paroxysmal attacks of pain referred to the gastric region and lower extremities, were necessarily considered of tabetic origin.

*Hysteria* and *epilepsy* claim a large proportion of these hypnotic conditions.

Lastly, after, as in the comas, careful, systematic clinical inquiry (history, physical examination, uranalysis, and investigations of the blood and cerebrospinal fluid) has been carried out and one of the foregoing hypnotic factors discovered, there remains a small number of unclassable cases for which must be preserved, until further light is shed on the subject, the term *essential* or *idiopathic narcolepsy*, probably of constitutional origin, encountered most often in neuropsychopathic families, and the most characteristic feature of which is that of being chronic, wholly incurable, and not accounted for by any clinically expressible cause

* * *

II.—**The lethargic states,** in the commonly accepted sense of the term, *i.e.*, states featured by morbidly prolonged sleep (persisting for days, weeks, months, or even years), and attended with pronounced slowing of the circulation and of nutrition, impose upon the practitioner two serious tasks

**1. In the presence of an actual lethargic state, to ascertain its nature.**

**2. In the presence of an actual state of death, to distinguish it from a lethargic state, and vice versa.**

Only exceptionally is a lethargic state attended with inhibition of the circulatory and respiratory functions to such an extent as to lead actually to apparent death as seen by a careful and trained observer. Ordinarily, indeed, an obscure grade of consciousness is still present and the taking of food is frequently possible. The lethal appearance, however, is the thing that proves striking to bystanders and is expressed in Ambrose Paré's definition: "Sleep by virtue of which the faculties and powers of the mind are buried, in such wise that it seems as if one were dead"

Death having been excluded by the findings later to be mentioned, the nature of the lethargic condition remains to be ascertained. Singular complications have been injected into the question by the doctrinal discussions concerning hysteria. Lethargy has been and is still considered by a few authors as a specific manifestation of hysteria; it has been termed, comatose hysteria,

*Hypersomnia* of the lethargic type, prolonged and profound, with pronounced slowing of the circulatory and respiratory functions

*Hypersomnia* accompanied by more or less definite symptom-groups:

Infectious states.
Meningeal conditions.
Toxic states.

*Hypersomnia* accompanied by motor manifestations.

I —**Hypersomnia presenting the appearances of normal sleep** and differing from it only in the depth or duration of sleep may be due to such a variety of causes—exhaustion, intoxications, infections—that its diagnostic significance is thereby greatly reduced.

The semeiology of profound sleep partially merges with that of the lethargic states (see below) and that of coma (see *Coma*). It will, therefore, be necessary here only to recall briefly that of **morbid somnolence or narcolepsy,** manifested in an abnormal and practically irresistible tendency to sleep, sometimes only of short duration, but at others almost permanent, as, *e.g.*, in the sleeping sickness.

This variety is met with in most of the *meningoencephalic states* (sleeping sickness, lethargic encephalitis, various meningeal disturbances, and brain tumor) (see below).

It is certainly caused oftenest by *toxic-infectious conditions,* which may be enumerated for purposes of memorization as follows:

*Heterotoxic hypnogenous intoxications:* Opium, chloral hydrate, alcohol, cannabis, chloroform, ether, and various hypnotics (barbital, sulphonethylmethane, etc )

*Autotoxic hypnogenous intoxications:* Acetonemia (diabetes), azotemia (uremia), ill-defined autointoxications due to hepatorenal insufficiency, intestinal fermentations, or slowing of tissue oxidation (*hyposphyxic states; cardiopulmonary insufficiency*).

Less frequently, these narcoleptic states are witnessed during the period of decline in *general infections;* some of them, such as the narcolepsies of convalescence, are actually of the nature of reparative sleep.

Perhaps the explanation of this anomalous occurrence is to be found in the combined action of cold and of the narcotic causing general vasomotor paralysis with marked reduction in the organic demand for oxygen—a condition similar to that of hibernation in certain animal species.

Was this a hysteric subject? The history does not mention it, but it is plain that the condition of lethargy and even apparent death was here brought about through the combined action of intoxication by a hypnotic and of exposure to cold

While undue weight is not to be laid on literary documentation, the case of Juliet—which is naturally brought to mind—seems to show that these toxicolethargic states were well known and had been accurately observed even before Shakespeare's time At all events, a more precise description of them would be hard to find:

> *. . . out, alas! she's cold,*
> *Her blood is settled, and her joints are stiff.*
> (*Romeo and Juliet*, Act IV, Sc. V).

> *Take thou this vial, being then in bed,*
> *And this distilled liquor drink thou off,*
> *When presently through all thy veins shall run*
> *A cold and drowsy humour, for no pulse*
> *Shall keep his native progress, but surcease*
> *No warmth, no breath, shall testify thou livest;*
> *The roses in thy lips and checks shall fade*
> *To paly ashes, thy eyes' windows fall,*
> *Like death, when he shuts up the day of life*
> *Each part, deprived of subtle government,*
> *Shall, stiff and stark and cold, appear like death·*
> *And in this borrow'd likeness of shrunk death*
> *Thou shalt continue two and forty hours,*
> *And then awake as from a pleasant sleep*
> (*Romeo and Juliet*, Act IV, Sc I).

Incidentally and without further discussion, the mysterious and disturbing spirit of Lazarus may here be alluded to.

The *deep sleep of convalescents* and of the *depressive form of melancholic stupor* rarely assumes actual lethargic characteristics, though a marked similarity may exist

In all such instances the diagnosis is indicated by the history far more than by direct observation of the case.

attacks of hysteric sleep, and hysteric apoplexy. While due account has to be taken of the possibility of simulation; while one should be on his guard, and while it is prudent to question, with Dupré and Meige, whether certain cases recorded and advertised in the public press are not "simply fantasies of mythomaniacs desirous of drawing attention to themselves," there seems to be no doubt but that *"hysteric sleep"* probably comprises the majority of the true lethargic states

Yet it does not appear to the author that lethargy is exclusively an attribute of hysteria. Lethargic states may seemingly be brought on by many other causes, *in particular through intoxication by hypnotics*, the resulting lethargic state being sometimes sufficiently profound even to induce a state of apparent death, as illustrated in the following case, recorded in the *Deutsche medizinische Wochenschrift* for November 13, 1919, page 1277. A nurse aged 23 took, on October 27, 1919, 1 7 grams of morphine and 5 grams of veronal. She was found the next day in a park, life being almost extinct. In the ambulance she was thought to be dead The existing signs of death were, rigidity, extreme pallor, and absence of reflexes, of the pulse, of breathing, and of the heart-beats. Application of hot sealing wax over the skin caused no reaction After she had been in the Morgue for fourteen hours, the coffin was opened on October 29, a medical official wishing to identify the body. The cheeks were seen to be slightly roseate and slight movements of the larynx were observed. No respiratory movements nor pulse beats were noted, but obscure heart sounds were audible At 10 o'clock in the morning the patient was taken to the hospital; hypodermic injections of caffeine and camphor in oil were administered and the stomach washed out. A hot bath was then given, with vigorous rubbing, followed by artificial respiration and oxygen inhalation At 11 o'clock the pulse was perceptible and a few brief inspiratory movements were noted; the rigidity of the limbs was passing off. At noon the pulse was distinctly noticeable, with a rate of 50 per minute On October 30, the patient regained consciousness and made short verbal statements She gradually recovered, but exhibited a persistent leucopenia.

In this case the arrest of the circulation and respiration with continuance of life appears to have lasted over twenty-four hours

*Mors certa, mors incerta,*
*Moriendum esse certum omnino.*
*Mortum esse incertum aliquando.*
(WINSLOW, 1740).

Thus, profound sleep, and especially certain morbid forms of sleep, may be confused, at least by hasty observers, with actual death. A few instances of unwarranted burial have been recorded, but the number of such cases has been singularly amplified through popular imagination. While such occurrences have doubtless been rather frequent in backward and somewhat savage countries, it should be repeatedly emphasized that very few authentic instances exist in civilized nations and that in Paris not a single case of this kind in the last thirty years has come to the author's notice. Yet the instance above referred to shows that such an occurrence is not an absolute impossibility

The fact remains, therefore, that in the case of the practitioner and more particularly the coroner's physician, professional conscience and the anxiety of relatives often place before him with singular force the question of apparent death and actual death. It is therefore appropriate to recall here the definite signs of death.

These indications are, on the whole, drawn just as they were a century ago, from the direct or indirect observation of permanent cessation of circulation, respiration, heat production, and the functions of the nervous system Modern observers have merely devised a few improved procedures which permit of ascertaining more accurately or more promptly the cessation of function of the systems referred to

A concise summary of these procedures, with brief descriptions of the technic, is given in the annexed table.

* * *

**III. Hypersomnia Associated With Definite Clinical Syndromes.**—Meningeal disturbances in which somnolence or sleep of varying depth and obstinacy is one of the chief features are extremely common. Coma is a usual ultimate manifestation

Special mention must be made, however, of three meningo-encephalic conditions, now rather well-defined clinically, and

**SIGNS OF DEATH DRAWN FROM:**

| A. Cessation of Circulation. | B. Cessation of Respiration and Heat Production. | C. Cessation of Function of the Nervous System. |
|---|---|---|
| *"Cor primum vivens, ultimum moriens"* (HALLER)<br>1. *Permanent cessation of the heart beats,* ascertained by prolonged auscultation (for 5 minutes) or twice at an interval of half an hour (Bouchut, Sommier)<br>2. Absence of golden yellow or orange color of the tissues and conjunctivæ after subcutaneous or intramuscular injection of 10 c c of a 1 5 alkaline solution of fluorescein, diffusion time 15 to 30 minutes (Icard)[1]<br>3. Acid reaction (to blue litmus) of the hepatic and splenic pulp collected by deep exploratory puncture (7 to 8 centimeters) If the litmus paper turns red, death is certain A test applicable thirty minutes after death (Ambard and Brissemoret)<br>4. Cessation of the pulse beats and absence of venous distention after ligature<br>5 Cadaveric hypostasis and lividity<br>6 Blistering test (Lorain, 1874) The flame of a candle is applied to the skin If person living *Blister due to burn* If dead *Dry elevation filled with air* (An inconstant and unreliable test.)<br>7. Forcipressure test (Icard) A portion of skin is strongly pinched with stout forceps If person living Traumatized tissues gradually return to their original appearance If dead: The depression persists indefinitely and the tissues assume a parchment-like appearance<br><br>[1] Icard's *formula*· Fluorescein, 10 grams; sodium carbonate, 15 grams; distilled water, 50 grams. | *"No warmth, no breath shall testify thou livest."* (SHAKESPEARE, *Romeo and Juliet.*)<br>1 *Permanent cessation of respiration,* observed by direct inspection, by absence of condensation of *moisture on a mirror, or* with a feather placed in front of the mouth and nostrils.<br>2 *Cadaveric cooling,* a progressive process, with eventual tendency to agreement with the external temperature after fifteen to twenty hours, cooling is gradual between 30° and 3°<br>3. *Cadaveric rigidity* Begins with the jaws and gradually extends to the lower extremities in 6 to 12 hours (*Jam Joetet*)<br>4 *Cadaveric putrefaction* Usually begins on the right side at the cecum and extends more or less rapidly to the whole lower portion of the abdomen (abdominal green discoloration).<br>"A piece of paper impregnated with a 1.4 solution of lead acetate and placed in front of the nostrils or lips of the subject presumed to be dead turns black owing to exhalation of sulphurous gases the result of putrefaction—especially pulmonary—already at the end of the first day." (Icard) | 1 Immobility, insensitiveness, muscular relaxation, and relaxation of the sphincters.<br>2 *Ocular signs.*<br>(*a*) Loss of the reflexes, especially pupillary and corneal.<br>(*b*) Reaction of the conjunctiva to ethylmorphine hydrochloride (dionin) A bit of dionin powder of the size of a wheat grain or 2 or 3 drops of a 1 3 solution causes an intense reaction with conjunctival chemosis in a living subject Absence of all reaction is a good indication of true death. Serviceable 2 hours after the supposed death (Terson).<br>(*c*) Subsidence and flaccid condition of the eyeball.<br>(*d*) Shiny film over the cornea<br>(*e*) Black spot on the sclera (on the outer side of the eye, with the lids half-open).<br><br>N B—*In a few instances in which anxious uncertainty on the part of relatives was extreme in spite of definite statements and the official certificate of the coroner's physician, the author did not hesitate to secure tangible proof of death by cutting a superficial artery (the radial).* |

3. *A condition of sleep, a hypersomnia,* ranging, as in trypanosomiasis, from simple somnolence to constant, deep, lethargic sleep, —without actual coma, however, as it is nearly always possible to rouse the patient.

As subsidiary manifestations one may note depressive states, paralysis of the extremities of hemiplegic type, sensory disturbances, pain, mental disturbances, or signs of excitement, such as muttering delirium, tremor, contractures, convulsions, and vasomotor disturbances.

*Examination of the cerebrospinal fluid* yields two important signs:

1. *Low cell content or absence of cells.*

2 *High sugar content,* 0 7 to 1 gram to the liter, probably due to stimulation of Claude Bernard's glycobulbar center

3. Exceptionally the cerebrospinal fluid may be hemorrhagic.

In truth, the pathogenic agent in lethargic encephalitis is as yet unknown, and its specificity not wholly demonstrated While the diagnosis is clinically obvious in cases combining typically the triad of symptoms already mentioned, it is far more difficult in the atypical, incomplete forms It seems wise, therefore, temporarily to maintain, with Claude, a clinical subdivision for:

**Meningeal conditions with narcolepsy,** in which the chief symptom is somnolence, present in association with fever, spinal rigidity, Kernig's sign, retention of urine, diminished reflexes, excess of albumin and lymphocytes in the cerebrospinal fluid, and sometimes headache, squint, and inequality of the pupils

Claude cautiously concludes "The concurrence of these narcolepsies attended by very pronounced meningeal reactions with an epidemic of influenza would seem to justify the view of some etiologic relationship between the two conditions"

As a matter of fact, narcolepsy may be met with in the majority of meningeal disorders, including *tuberculous meningitis* in particular

**Brain tumor** is associated with insomnia much more frequently than with narcolepsy, which, in any case, is here of but slight diagnostic service and of no localizing value.

constituting actual examples of a narcoleptic meningo-encephalitis, *viz.*, the sleeping sickness, lethargic encephalitis, and post-influenzal meningeal disorders with narcolepsy.

The true sleeping sickness is, as is well known, endemic in the African territories near the equator, and is specifically caused by primary development in the blood and secondary development in the cerebrospinal fluid of the parasite known as *Trypanosoma gambiense*. In its primary stage of blood infection, clinically manifested in trypanosomic fever and glandular enlargement, somnolence is absent or slight, and the disturbance might be mistaken for malaria or filariasis; under these conditions blood examination is necessary, and will remove all doubt by revealing either the trypanosome, plasmodium, or filaria.

In its secondary or cerebrospinal stage, somnolence becomes pronounced. In the waking state the patient is confused, sleepy, with drooping lids, a dull expression, sluggish, benumbed mentality, and an unsteady gait. Somnolence is invincible, irresistible, and permanent; sleep is constant or nearly so, but is relatively light, the patient being easily roused by the slightest sound. *Fever, weakness, tremor, asthenia, emaciation, and progressive* cachexia complete the clinical picture. If the diagnosis was not confirmed by blood examination in the preceding stage, examination of the cerebrospinal fluid now clinches it by revealing the trypanosome.

Lethargic encephalitis has been the subject of a large mass of literature in the last few years, whence it appears that the condition is characterized by three main symptoms:

1. *Fever,* 39° to 40° C., associated with weakness and loss of weight

2. Dissociated *ocular paralyses,* sometimes combined with *facial paralysis.* This would appear to be, perhaps, the most constant and striking manifestation of the disease, the patients frequently seeking medical advice because of seeing double (diplopia), because of squint (strabismus), or because the lids fail to stay open (ptosis). Upon examination there may be observed inequality of the pupils, paralysis of accommodation, ptosis, and dissociated paralyses of the motor nerves resulting in disturbed movements of the eyeballs.

ambulistic conditions inquiry should be made for some *obsession, intoxication,* or a *neuropsychopathic predisposition* It seems difficult to set up an absolute line of demarcation between true somnambulism and the clear-cut, active dream-states in which insane illusion, continuing for a time "after the awakening," leads the subject to carry out unconscious, absurd motor acts.

Attacks of MAJOR SOMNAMBULISM, featured by the duration and complexity of the motor acts carried out, or by their repetition, are of more definite clinical significance They are hardly met with in any conditions other than *epilepsy* and *hysteria,* and according to some authors in *neurasthenia* and *alcoholism* The forms encountered are sometimes sufficiently distinct to permit of the making of a differential diagnosis.

*Epileptic somnambulism* (and fugue, or wandering tendencies) is characterized sometimes by sudden onset, imperious, blind impulses, the absence of any definite purpose, and complete amnesia

*Hysteric somnambulism* (and fugue) frequently reflects the unconscious influence of a previous idea, whence there results some degree of logical coherence of motor acts directed to some more or less definite purpose, a vague consciousness of which may persist after awakening

*Neurasthenic,* or better, psychasthenic *somnambulism* (and fugue), the occurrence of which is doubted by some writers, is claimed to be associated with relative consciousness of the obsessive idea, logical sequence in the motor acts carried out, and almost complete remembrance upon awakening

*Alcoholic somnambulism* is described as usually occurring only among congenitally psychopathic subjects, and as exhibiting the features of epileptic or neurasthenic somnambulism, according to the individual case.

Brief mention may here be made of a condition of limited clinical diagnostic interest, *viz., somnambulism induced during hypnosis,* which has been and still is the subject of acrimonious discussions This practice obviously serves the purposes of therapeusis rather than of diagnosis.

* * *

It has always been a matter of surprise to the author, and particularly so while writing this section, that most of the terms

**IV. Morbid Sleep Associated With Motor Manifestations.** —This subject is highly complex and covers a singularly large field, as it brings up the question of ambulatory automatism, somnambulism, and the cataleptic states; leads, without undue extension, to the interpretation of insane delusions and wandering tendencies, to which it bears an obvious connection, and brings up in last analysis the vexing problems of hypnotic suggestion, dual personality, and responsibility of the human individual.

One need here merely point out the interrelationship of these questions and briefly recall the clinical significance of somnambulism, which is obviously inseparable from *ambulatory automatism.* The definition, limits, and meaning of the term somnambulism have been rendered singularly vague through the conflicts between different schools of thought concerning the nature and even the existence of hysteria From the standpoint of elementary clinical common sense, *somnambulism* is constituted of the fact that a sleeping person may carry out more or less complex motor acts of which he loses all remembrance upon awakening.

The MINOR SOMNAMBULISTIC STATES are commonly observed conditions and may be witnessed in practically normal subjects under the influence of some rather compelling thought or mild intoxication; constitutional neuropsychopathic states predispose to these manifestations. Their frequency is surprising if they are looked for at all carefully. A person with simple nervousness, not described by any definite clinical term, after a rather strenuous and difficult voyage, during which a single obsessive thought had kept him almost awake for a whole week, and in which, as he bore with him a rather large sum of money, he had naturally had to take certain precautions against being robbed, was led for months to brief somnambulistic practices which consisted in his getting up to see that the door of his room was closed and his wallet safe in his inner coat pocket. The obsessive thought by which he had been dominated for a whole week left an impression that wore off only after several months

Alcoholic somnambulism, met with especially in persons with inherited psychopathic taint, is to be compared with the alcoholic delusional state known as *delirium tremens.* In these minor somn-

Λήθαργος, as adjective, one who is somnolent; as noun, the desire to sleep; from λήθη, forgetfulness, without exact knowledge of the mode of derivation

Common acceptance of the term.

1. A state of apparent death characterized by almost complete suspension of respiration and circulation.

2 Profound, irresistible, uninterrupted sleep, lasting several hours or days.

**Narcosis.**

Stupor due to the action of a narcotic.
*Derivation:* Ναρκωτικός, from νάρκη, stupor

**Narcotic.**

That which produces stupor, like opium, hyoscyamus, belladonna, etc.

**Narcotism.**

The aggregate of the effects caused by narcotic substances Same derivation

**Narcotin.**

A chemical term An alkaloid discovered in opium by Derosne in 1803; also known as Derosne's salt.
*Derivation:* Narcotic.

**Narcotico-acrid.**

An adjective and toxicologic term A word applied to the poisons which, like aconite, helleborus, etc., induce both narcotism and inflammatory effects in the intestine.
*Derivative words* Narcolepsy, narcomania, etc

**Oneiric.**

From ὄνειρος, a dream Something in the nature of a dream Oneiric delirium Dream-like delirium
*Derivative words* Oneirocritia (the art of interpreting dreams), oneiromancy (divination or diagnosis through dreams)

**Sleep.**

Complete stupor of the senses, or, in physiologic terms, a temporary cessation of the activity inherent in animal life

**Somnifacient.**

Something which induces sleep
*Derivation* From the Latin, *somnus,* sleep, and *facere,* to make

**Somniloquy** (somniloquence).

The habit of talking during sleep
*Derivation:* From the Latin, *somnus,* sleep, and *loqui,* to speak.

**Somnolence.**

Slight drowsiness which is unpleasant but irresistible.
*Derivation:* Latin, *somnolentia,* from *somnus*

**Somnolent.**

Affected with somnolence
*Derivation:* Latin, *somnolentus,* from *somnus.*

relating to sleep, and especially its abnormal forms, are so vague and ill-defined in their meanings. Thus, the terms *catalepsy, lethargy,* and *somnambulism* each have two entirely different accepted meanings, popular and scientific, and even the latter is far from being uniform as used by different writers It has seemed indispensable, therefore, to prepare a short glossary of hypnotic terms:

**Catalepsy.**

1. A disorder featured by the ability of the limbs, and even the trunk, to maintain throughout the attack positions which they had previously occupied or had been placed in

2. A condition in the presence of which the subject is unable to move, although consciousness and sensation are retained

[Historical (16th century) "And if said vapors ascend to the brain, they cause epilepsy or catalepsy, which is when the whole body remains stiff and cold and in the same attitude which it was in before the illness, the eyes being open without seeing and without hearing" (Ambrose Paré, xviii, 52.)].

*Derivation* Κατάληψις, from κατά, down, and λῆψις, seizure

**Dream.**

An involuntary combination of images or ideas, generally without sequence, sometimes very distinct and well co-ordinated, occurring during sleep

**Hypnosis.**

Commonly Artificial sleep Example· Chloroform hypnosis [Also abnormal sleep, and the approach or production of sleep.]

*Derivation* Ὕπνος, sleep

**Hypnagogue.**

Something which leads to sleep Hypnagogue hallucinations Visions experienced when half asleep

*Derivation* Ὕπνος, sleep, and ἀγωγός, leading

**Hypnology.**

The study of sleep λόγος, treatise.

**Hypnophobia.**

Fear of sleeping φόβος, fear or terror during sleep

*Derivative words* Hypnotic, hypnotism, and hypnolepsy.

**Lethargy.**

Deep and continued sleep in which the patient will talk if awakened but does not know what he says, forgets what he has said, and sinks again into slumber.

*Derivation* ληθαργία, λήθαργος, see *Lethargus*

**Lethargus**

A term applied by the old Greek physicians to a remittent type of fever featured by drowsiness.

## SLOW PULSE (BRADYCARDIA).

[*βραδύς, slow, καρδια, heart; slowing of the heart.*]

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True bradycardia is attended by a slowing of the rate of cardiac contraction to 56 per minute or lower. Palpation of the

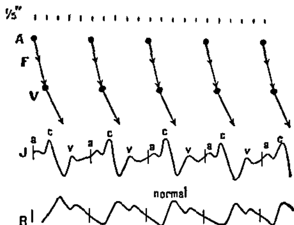



Fig 862—Normal heart-rhythm

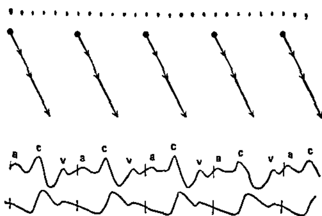



Fig 863—Tendency to auriculoventricular dissociation Delayed conduction Prolongation of the *a-c* period

pulse in combination with auscultation of the heart is essential for the recognition of true bradycardia Pseudo-bradycardia, or bradysphygmia, *i e.,* slowing of the pulse rate without actual

**Somnambulism.**

1. A disorder of the cerebral functions featured by a tendency to repeat during sleep acts that have become customary, or of walking and carrying out various motor acts without retaining any memory of them on awakening

2 Magnetic somnambulism. A special nervous state into which highly susceptible persons, especially hysteric women, can be thrown by a species of psychic influence.

3 Commonly accepted meaning A nervous state during which a sleeping person rises from bed and carries out certain motor acts which he fails to remember on awakening

*Derivation* Latin, *somnus*, sleep, and *ambulare*, to walk.

**Soporific.**

Something which induces profound sleep

*Derivation* Latin, *soporificus*, from *sopor*, sleep.

not literally valid and that rather numerous ambiguous or transitional conditions occur, the subjoined synoptic table is available for practical purposes.

## DIFFERENT TYPES OF BRADYCARDIA.

| Clinical Features | Graphic Features | Various Reactions | History |
|---|---|---|---|
| **Due to Intracardiac Disease.** Disease of the bundle of His (gumma) Ventricular myocardial disease (uncommon) (fibrous or rheumatic myocarditis). | | | |
| Bradycardia. 1 Very pronounced (30 or 40 beats per minute, or less). 2. Continuous or paroxysmal. | Complete independence of the auricular beats, the rate of which remains practically normal, from the ventricular beats, with their markedly reduced rate (Heart-block Auriculo-ventricular dissociation) | No reaction to atropine No reaction to the respiratory movements, to exertion and posture nor to fever | Specific history most frequently Sometimes combined jaundice and uremia During the initial period (paroxysmal bradycardia), severe and prolonged epileptoid seizures (Stokes-Adams disease). |
| **Due to Pathologic Change or Functional Defect in the Vagus** | | | |
| Bradycardia. 1 Moderate: 56 to 40 2 Transient. | Usually a total bradycardia without auriculo-ventricular dissociation. | Marked reaction (acceleration) to atropine Positive reaction (acceleration) to motion, posture, respiration, exertion, and fever | Nervous depression: Lassitude, exhaustion, shock, neurasthenia, and psychoses Hepatic. Jaundice Pharmaceutic Strophanthus, digitalis. Autointoxication: Uremia. Nervous lesions involving Medullary center: Hemorrhages, softening, arteriosclerosis, etc. Points of emergence: Meningitis Nerve-trunk: Enlarged tracheobronchial glands, mediastinal tumors, or aneurysm |

slowing of the heart rate, may occur in the alternating pulse and in the bigeminal pulse of extra-systole when the second pulsation is too weak to be perceptible at the wrist; under these conditions

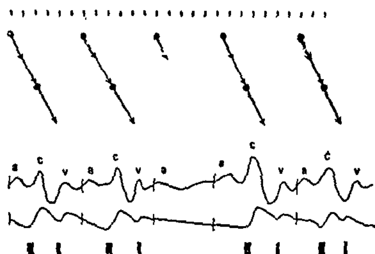



Fig. 864.—Partial heart-block. Incomplete auriculoventricular dissociation.

*only one pulsation is felt for every two beats of the heart (see Arhythmia).*

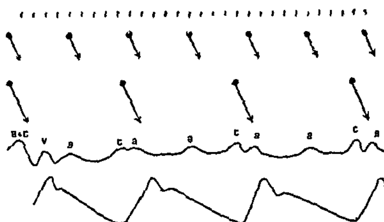



*Fig. 865.—Complete auriculoventricular dissociation. Co-ordination between the contractions of the auricles and ventricles is wholly lost.*

Although recent investigations, such as those of Daniel Routier, of Frédériq, and Petzetakis, in particular, have shown that the accepted division of bradycardia into several varieties is

The atropine test consists in injecting into the bradycardic subject 0.001 gram of atropine sulphate (1 cubic centimeter of a 1:1000 solution), and later, in the succeeding days, 2 cubic centimeters if the first test was negative and well borne

The test is considered negative if, in the hour following the injection, acceleration by less than ten beats per minute occurs

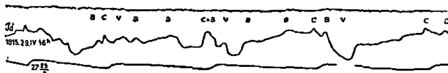


Fig. 870.—Bradycardia due to auriculoventricular dissociation the result of a gumma of the bundle of His

Case 1257. H, 1855, pulse, 27, pressures, 25/9. Former specific infection Wassermann +

The test is considered positive if, in the hour following injection, acceleration greater than twenty beats per minute occurs.

The test is considered doubtful if, in the hour following injection, an acceleration of between ten and twenty beats occurs

It is held that any bradycardia which is not changed in the atropine test cannot be considered a nervous bradycardia. Atropine, termed by François-Franck "the curare of the nerves of the heart," prevents the inhibitory, bradycardic action of the vagus. This rule, however, is still under widespread discussion.

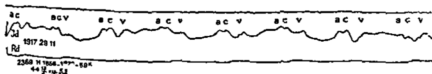


Fig. 871.—Total bradycardia due to depressive psychoneurosis

In typhoid fever, Lutembacher has sometimes observed a paradoxic slowing of the pulse after injection of 0.0015 gram of atropine This is because, when the sinus is but slightly inhibited, the atropine, annulling vagal inhibition, accelerates the sinus rate, this in turn abruptly aggravating the disturbance of conduction.

The above schematic classification is clinically convenient, but there are still many incompletely elucidated points.

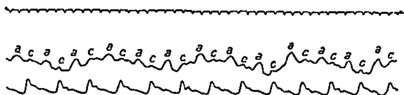


Fig 866—Delayed conduction (*Routier*).

1. **Anatomic:** There exist auriculo-ventricular bundles independent of the bundle of His (Stanley Kent).

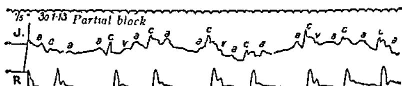



Fig 867—Partial heart-block (*Routier*)

2. **Physiologic:** Persistence of auriculo-ventricular conduction after section of the bundle of His (Stanley Kent).

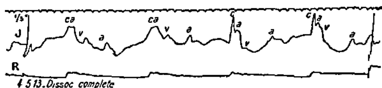



Fig 868—Complete dissociation (*Routier*)

3. **Physiopathologic:** (*a*) Removal of block from some hearts by adrenalin (D. Routier).

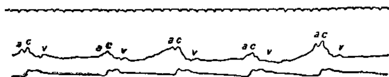


Fig 869—Total bradycardia (*Routier*).

(*b*) Block in some hearts upon compression of the eyeballs (Petzetakis). In total bradycardia of nervous origin, compression of the eyeballs (*oculocardiac reflex*) may result in ventricular automatism

Second stage: Ulcers with punched-out margins encroaching upon the pillar and digging into the tonsil, the base containing a pseudomembranous exudate; no induration.

(1) Bacteriologic examination, especially necessary in the pseudomembranous and ulceromembranous forms (see *Bacteriology*); (2) the associated clinical signs, and (3) the visible features just referred to, allow, as shown in the subjoined table, of a rather thorough and clinically serviceable discrimination between the various forms of acute angina.

The mistaking of a non-diphtheritic pseudomembranous sore throat for diphtheria entails but little detriment to the patient, especially in children, as it leads to the injection of serum, which generally acts favorably even in non-diphtheritic sore throat.

The opposite error, *i e.*, that of overlooking diphtheria, is a pernicious one It should be thoroughly realized that there occur atypical forms of diphtheritic sore throat, clinical diagnosis of which is difficult, *e g.*, the catarrhal and pultaceous forms, the crypto-diphtherias, and the herpetic, follicular, and phlegmonous forms In this connection the practical rules of Darré are worth remembering:

Any sore throat occurring in an epidemic should be regarded as suspicious, whatever be its clinical aspect. Any sore throat accompanied by rhinitis and laryngitis is probably diphtheritic, or at least should be treated as such.

Any sore throat affecting successively several members of the same family is rather likely to be diphtheritic, and should be investigated most carefully. In respect of diphtheria, when there is doubt abstention is not permissible, mere clinical presumption is sufficient to demand serum injection, at least in children, without waiting for the result of bacteriologic examination.

Lastly, reference may be made to the probably unsuspected frequency of chronic or subacute pharyngeal infections as the underlying cause of "cryptogenic" fevers, refractory rheumatoid manifestations of undetermined nature, and of inveterate, vague deficiencies in the general condition of the body. The pharynx and nasopharynx certainly constitute one of the great foci of systemic infection. They should always be subjected to examination, especially in the presence of subacute or chronic febrile states and erratic joint troubles of obscure origin.

## SORE THROAT (ANGINA).[1]

[Angina, *from* angere, to *suffocate.*]

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Acute sore throat may be **primary**, *i.e.*, occur as the outward evidence of an infection involving the pharynx first, or secondary, *i.e.*, occur as the pharyngeal manifestation of a more general infection, whether constituting its first or its last symptom.

Whether *primary or secondary*, sore throat appears either in the form of a **red throat** (red angina), with more or less diffuse and intense redness of the tonsils and pharynx, without white patches;

Or in the form of a **white throat** (white angina), with the presence of whitish or grayish exudates, which vary in conspicuousness according to the degree of contrast with the red background of inflamed pharyngeal mucous membrane.

Such white anginas occur in the following four types:

(*a*) **Pultaceous.**—The tonsil shows disseminated white points or is covered with a grayish, creamy, puriform, friable, non-adherent exudate which can be detached merely by contact of the tongue depressor or of cotton on an applicator.

(*b*) **Vesicular.**—The pharynx, soft palate, and tonsils are at first the seat of a more or less extensive and confluent vesicular eruption, rupture of which results in the formation of small circular ulcerations or small patches of false membrane.

(*c*) **Pseudomembranous.**—The pharynx, red and swollen, is covered with grayish yellow false membrane which is adherent to the mucous membrane, thick, detached only with difficulty, causing the mucous membrane to bleed, and recurring more or less rapidly after removal.

(*d*) **Ulceromembranous.**—This type runs a two-stage course:

First stage: False membrane on the tonsil, whitish, only slightly adherent, and readily reproduced.

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[1] [illegible] that is both rational and [illegible] outlines based on but a single feature will stand the test of clinical usage; all one can do is to combine in the least objectionable manner the clinical and bacteriologic data as well as those relating to the course of the disease. The plan of division herein followed, now in rather general use, is one of the least defective in the present state of our knowledge.

Second stage: Ulcers with punched-out margins encroaching upon the pillar and digging into the tonsil, the base containing a pseudomembranous exudate; no induration.

(1) Bacteriologic examination, especially necessary in the pseudomembranous and ulceromembranous forms (see *Bacteriology*); (2) the associated clinical signs, and (3) the visible features just referred to, allow, as shown in the subjoined table, of a rather thorough and clinically serviceable discrimination between the various forms of acute angina.

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| General Features | Associated Clinical Signs | Bacteriologic Examination | Primary and Secondary. |
|---|---|---|---|
| | | **Red Throats (Red Angina).** | |
| Erythematous.<br>Varying degree of redness of the tonsils and pharynx. | Dysphagia Fever.<br>1 Catarrhal, resolution within a few days<br>2 Recurrent<br>3 Phlegmonous, ending in abscess formation | Primary No specific germs (streptococci, pneumococci, or staphylococci)<br>Secondary· Frequently streptococci and associated micro-organisms. | Primary.<br>Secondary:<br>Scarlet fever: Initial sore throat; an intense erythema, often pre-eruptive<br>Measles Initial sore throat; red dotted appearance, often pre-eruptive.<br>Erysipelas· Erythema, then blebs, preceding or following facial erysipelas |
| | | **White Throats (White Angina).** | |
| Vesicular.<br>*Herpetic.*<br>1st stage: Vesicular.<br>2d stage: Pseudo-membranous, with polycyclic outlines. | Abrupt onset.<br>Dysphagia<br>1 Sometimes herpes on the lips, cheeks, etc<br>2 *Sometimes recurring* pharyngeal herpes (investigate as regards abuse of tobacco or syphilis) | As in red throats. | Primary:<br>1 Pharyngeal herpes zoster.<br>2 Primary herpetic angina<br>Secondary:<br>1 Herpetic angina secondary to other manifestations of herpes<br>2. Recurring pharyngeal herpes; investigate for tobacco abuse or syphilis.<br>3 Aphthous angina following stomatitis |
| Pultaceous.<br>Whitish, creamy, non-adherent exudates. | Dysphagia Fever<br>1 *Follicular* (lacunar or cryptic), resolution in a few days<br>2 Recurrent<br>3. Phlegmonous. Follicular (lacunar) abscess | As in red throats. | |

| Pseudomembranous Angina.—A Diphtheritic. | | | |
|---|---|---|---|
| **Pseudomembranous.** **Light grayish, thick, and adherent exudates.** | *Dysphagia.*<br>Glandular enlargement.<br>*Fever*<br>Evidences of severe *systemic intoxication.* | The Klebs-Löffler bacillus in more or less pure culture or in varying admixture with the germs mentioned below is found in smears and particularly in cultures (see *Bacteriology*). | **Primary:** Diphtheritic. |
| **B. Non-diphtheritic.** | | | |
| | Special attention paid to the usual absence of fever and of evidences of systemic intoxication in syphilitic manifestations | Micrococci: *Generally mild; no evidences of intoxication, albuminuria, nor glandular enlargement.*<br>Staphylococci } Relatively mild<br>Pneumococci }<br>Streptococci Usually serious.<br>These germs may be present in combination Combinations including streptococci are particularly virulent.<br>Syphilitic: Spirochetes. | **Secondary:**<br>Scarlet fever: Late angina, *sometimes severe, frequently* with associated diphtheria and streptococci<br>Small-pox and chicken-pox.<br>Typhoid fever.<br>Syphilis<br>1 Syphilitic chancre: *Cartilaginous induration* and marked post-maxillary glandular enlargement (see *Glandular enlargements*)<br>(Little or no febrile reaction)<br>2 Mucous patches<br>Tonsillectomy. |
| **Ulceromembranous.**<br>1. Pseudomembranous exudate.<br>2. Round or oval ulceration. | Dysphagia<br>Salivation<br>Malodorous breath<br>Glandular enlargement.<br>No induration. | Primarily<br>Vincent's fusiform bacillus and spirochete<br>As added infection<br>Pyogenic organisms<br>Pneumococci.<br>Colon bacilli | **Primary:** Vincent's angina<br>**Secondary:**<br>Tuberculosis of the tonsil (ulcers)<br>Easily distinguished from the preceding, thus<br>1 Preceded by miliary tubercles<br>2 Little or no glandular enlargement<br>3 Ulcer margined with yellowish granulations<br>4 No fusiform bacilli nor spirochetes in the exudate<br>5 Obvious former visceral tuberculosis |

## TINNITUS AURIUM. [Tinnitus, "*a tinkling.*"]

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"*De minimis non curat praetor*" is a well known adage. It would be unwise, however, to apply it to the *minor symptom known as* **tinnitus aurium**, or ringing in the ears While sometimes really of negligible clinical significance, it may, on the other hand, be an indication of previously unsuspected morbid conditions. When persistent, it becomes to the patient an unbearable obsession, which may lead to what amounts practically to a delirium.

*In a patient who comes complaining of distressing and obstinate* tinnitus, whether in the form of whistling sounds, cockle-shell sounds, or buzzing sounds, the proper procedure is to examine **first of all the ear** and make a systematic search for any lesion of the external, middle, or internal ear which might be the cause of the tinnitus.

Tinnitus due to **quinine**, subjectively resembling the ringing of bells and accompanied by hardness of hearing and a heavy feeling in the head is practically a normal condition which passes off completely in a few hours It occurs so constantly after quinine that it serves, in a measure, as a control of quinine exhibition. Where it is lacking the physician should **carefully inquire**: 1. *As to whether the patient has actually taken the dose prescribed.* 2. *Whether the salt supplied was actually the salt ordered.* 3 *Whether the medicine was taken in the manner specified* (with an acid beverage).

When intense, these auricular disturbances may become abnormal and alarming, or even progress toward a highly severe vertigo comparable to that of Ménière's disease According to Lermoyez, "all depends on the condition of the labyrinth, and if there is the least labyrinthine lesion, the least dose of drug is sufficient to produce manifestations of vertigo," and the administration of quinine is contraindicated.

Such marked disturbances are seldom induced by *sodium salicylate;* yet one should take into account the possibility of a tem-

porary brain congestion with tinnitus, transient deafness, slight dizziness, flushing of the face, and even epistaxis.

The frequency of tinnitus aurium in chronic aortitis and arteriosclerosis is well known; it may even induce giddiness or fainting spells. It is often a preliminary indication of further trouble to come. In any elderly patient complaining of tinnitus and dizziness a careful examination of the cardiovascular system and kidneys should be made, including auscultation, blood-pressure determinations, and uranalysis. The pathogenesis is com-

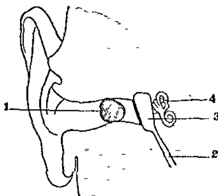



Fig. 872—Mode of production of tinnitus aurium (*G Laurens*).

Tinnitus is the result of irritation of the internal ear The latter may be due, in turn, to a disturbance (*a*) Of the external ear (wax), exerting pressure on the drumhead, ossicles, and endolabyrinthine fluid (1). (*b*) Of the middle ear (3), *e g*, otitis or catarrh or disease of the Eustachian tube (2), in the same way as the preceding condition (*c*) Of the internal ear (4), *e g*, disturbed circulation, anemia, congestion, constitutional disorders, arteriosclerosis, etc.

plex, but the condition is probably dependent largely upon reflex spasm of the capillaries in the brain, probably due, in turn, to the irritation to which the intima of the aorta is subjected through changes in the blood-pressure caused in aortic and arteriosclerotic cases by changes of posture, locomotion, and exertion.

Tinnitus aurium and vertigo are very closely allied (see *Vertigo*).

## Usual Causes of Tinnitus Aurium.

**External ear:** *Impacted cerumen or a foreign body.*

**Middle ear:** *Otitis media, especially with exudation and pressure on the stapes, or obstruction of the Eustachian tube.*

**Internal ear:** *The exciting cause of tinnitus is always actually some form of irritation of the internal ear, some disturbance in its circulation (hyperemia or ischemia), or an increase or decrease of intralabyrinthine pressure.*

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**In the absence of any apparent disease of the internal or middle ear:** An examination should be made for *degeneration of vessels, diathetic disorders,* and *pharmaceutic* or *autogenous toxic states.* These conditions induce tinnitus through the hyperemia or ischemia, or the rise or fall of pressure which they cause in the internal ear.

The following conditions should be looked for

**Arteriorenal degenerative changes:** Arteriosclerosis or Bright's disease

**Diathetic disorders of the neuroarthritic type:** Gout or diabetes

**Drug intoxications:** Quinine or sodium salicylate

# TONGUE, DIAGNOSTIC FEATURES RELATING TO THE.

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The tongue has been aptly said to "mirror the stomach," but it also supplies a clue to the functional state of many other structures, when inspected by a practised eye

**I. Examination of the Tongue Reveals Neither Ulcerations nor Tumor-like Formations.**—Aside from all malformations, ulcerations, and tumors of the tongue, to which brief reference will be made later, the appearance of the lingual mucous membrane *is in itself highly informative* The normal roseate and moist appearance, with the characteristic leaf-like oblique striations and the posteriorly situated V-shaped angle, are too familiar to require mention. It may be pointed out, however, that even under physiologic conditions the lingual mucous membrane is frequently covered with a *light grayish normal coating* resulting from the continuous desquamation of the superficial epithelia This coating accumulates as the interval since the preceding meal increases, mastication of solid food, on the other hand, is generally sufficient to cause it to disappear. One may therefore expect to find this coating thicker and more persistent in persons on a diet, taking liquid food, as well as in the morning on awakening. The presence of such a coating in the morning obsesses some patients to such an extent that they scrape their tongues every morning with some scraping instrument

On the other hand, *in sialorrhea the tongue is red, moist, and glistening;* it is, in fact, too clean-looking, and should lead to the discovery of excessive salivation, the cause of which is thereupon to be inquired into.

With the practically normal coating is combined an *almost rasp-like dryness* in heavy smokers and especially in mouth-breathers (coryza or adenoid vegetations)

The whitish or grayish color of the coating may be changed *as a result of the ingestion of certain foods or drugs* It may turn to a

color ranging from purplish red to dark brown upon ingestion of red wine, of blackberries, of chocolate, of extract of licorice, of catechu, of cinchona, of fresh nuts, of tobacco, etc.; it becomes saffron yellow upon ingestion of laudanum or rhubarb, and creamy white in persons on a milk diet.

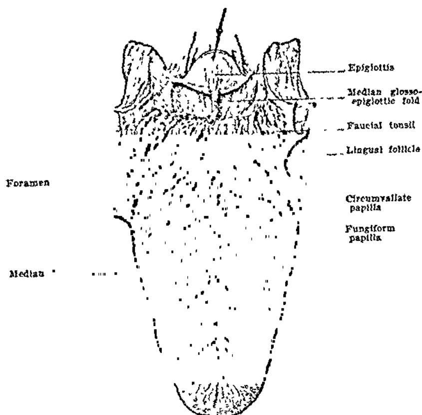



Fig 873—Dorsal surface of the relaxed tongue (*Poirier*).

Certain peculiarities the result of defective functioning of the digestive tract are universally known, in particular the dirty yellow brown coating of dyspepsia and indigestion. In gastric disorders the brown coating is thick and the tongue flattened out, with its margins showing imprints of the teeth, sometimes with small red elevations, consisting of the papillæ, when clearing begins.

Some infectious diseases affect the tongue in almost pathognomonic fashion:

The "strawberry" or "raspberry," scarlet red, bare tongue of *scarlet fever.*

The varnished, porcelain-like tongue, with a red band at its margins, of *grippe at its height.*

The parched, dry, fissured tongue, covered with dry and blackened mucus, of the *typhoid patient* (when poorly cared for, one might add, *i.e.*, where the ordinary measures of oral hygiene are not carried out).

The "sooty," dry, black, rough, "parrot tongue" of grave infections of the *typhoid type* and of *certain varieties of uremia.*

The phrase "certain varieties" is used in reference to uremia because in some other forms, in particular in certain instances of *acute azotemia,* the author has had occasion to observe a *thick, "viscous"* tongue, with excessive flow of viscid saliva containing a large amount of urea.

II. **True glossitis is reached by insensible gradations from the preceding group.**

1. **Thrush** is the most frequent disorder in *infancy,* being a result of poor quality of the milk or unclean bottles In the adult and elderly, it may occur as a complication of *grave infectious states* and of *cachexias,* as in advanced tuberculosis, severe typhoid fever, infections of the urinary tract, etc The tongue, at first red and "varnished," becomes covered with small grayish masses like milk curds, which thereupon draw together, fuse, and expand into creamy patches which can be detached rather easily by rubbing

2. In the **mercurial stomatitis** of patients intolerant of mercury and ignorant of oral hygiene, the tongue is swollen, viscous, of a dirty gray color, and bearing imprints of the teeth on its margins. The malodorous breath, the ptyalism, the coexisting stomatitis, and the history of having taken mercury lead directly to the diagnosis.

3. **Aphthous fever** is, as a rule, rather discretely manifested on the tongue. *Aphthæ* may be noted at the tip and along the margins of the tongue in the form of vesicles similar to those of skin herpes or of a circle of swollen mucous membrane; the vesicles rupture after a few days, leaving small circular ulcers of the size of a pin-

head up to that of a lentil, with grayish bases and irregular margins.

4. **Marginal exfoliative glossitis** is a chronic, recurrent affection of the tongue characterized by desquamation of plate-like scales with circinate, "geographic" margins, usually with their convexity directed outward.

The very multiplicity of the terms applied, *viz.,* pityriasis linguae (Rayer), geographic tongue (Bergeron), marginal exfoliative glossitis (Fournier), eczéma desquamatif lingual en aires, marginé (Besnier), psoriasis linguae, etc., is sufficient proof of our ignorance of its nature—which may perhaps be variable and complex. It does not seem to be of syphilitic origin; at least it is not amenable to specific treatment. Yet, as in the case of leukoplakia, to be next discussed, the distinction to be made from syphilitic glossitis is most difficult, and how many dermatologists have—as of yore in connection with tabes and general paralysis—used the phrase "parasyphilitic manifestation" in this connection? The "What do I know?" attitude of Montaigne is alone justifiable at the present time.

5. **Leukoplakia buccalis** appears in the form of amorphous, pearl-white, patches on the tongue, without any special marginal band; the patches are irregularly dispersed, thin, bluish, translucent, discrete at first, and show an almost invincible tendency to extend, thicken, and undergo hardening, with resulting formation of whitish, thick, adherent patches, which become fissured and cracked through desquamation. Such patches may also be observed on the mucous membrane of the lips and on the inner surface of the cheeks.

Syphilis is noted in a very great many of these cases, and it seems likely that a large number of leukoplakias are of syphilitic origin. Yet such exciting, provocative, or predisposing causes as tobacco (nicotinic leukoplakia), traumatism (dental leukoplakia, with dental caries or poorly made dentures), neuroarthritism (irritative neuroarthritic leukoplakia of Brocq), etc., play a rôle in their production which is not negligible.

The marked clinical interest of leukoplakia resides in its relationship to syphilitic glossitis and the possibility of the occurrence in it of epitheliomatous degeneration.

Brocq, with his usual ability, discussed this question, which is of prime clinical importance, in 1919.[1] The following three points are worth quoting here:

(*a*) There occurs a *buccal lichen planus* which is sometimes hard to distinguish from other conditions. It is marked by the presence, on the inner aspect of the cheeks, of white striæ exhibiting occasional small nodular elevations; on the tongue one observes instead opalescent spots, which coalesce to form more extensive patches.

(*b*) Leukoplakia *nearly always* develops on a syphilitic substrate, *but not always Non-syphilitic leukoplakias do occur.*

These two propositions are of marked practical import, for while they justify antisyphilitic treatment in the majority of cases, they condemn the blind tendency to institute intensive and repeated antisyphilitic treatments in all persons with ordinary white patches In playing the part of the wise and well-versed clinician, it is well to make a careful diagnosis in these cases (discrimination from lichen planus, or syphilis—manifest, probable, or non-existent) and to act accordingly as regards treatment.

(*c*) It is certain that many leukoplakias undergo epitheliomatous degeneration. "Yet, many subjects harboring leukoplakial patches do not show degeneration of the disorder into cancer; such degeneration of the disorder is even relatively rare when the patients, warned in good time, carry out and persistently continue all the required hygienic measures" (Brocq, *loc. cit*)

6 **Syphilitic glossitis** is of greater importance in the adult than any of the preceding varieties It is well established, to be sure, that the majority of instances of *leukoplakia* and a few instances of *marginal exfoliative glossitis* are syphilitic.

Syphilis may yield lingual manifestations in any of its stages: Primary (chancre of the tongue); secondary (various forms of secondary glossitis, mucous patches); tertiary (gummas, tertiary syphilomas).

Some varieties are merely exudative and exfoliative, like the majority of the secondary manifestations; others are tumor-

[1] Brocq: *Presse méd*, May 22, 1919.

producing and ulcer-forming (gummas). They consequently occupy a border-line position in the didactic classification herein followed. Yet they will be placed in the present group. They serve for purposes of transition to the clinical cases of glossitis associated with ulcers or tumor-formations.

**Chancre of the tongue** is relatively uncommon, representing hardly more than 10 per cent of the cephalic chancres. It is generally located at the tip of the tongue, and exhibits either an erosive, papulo-erosive, or ulcerative character with diffuse induration. The submaxillary lymph-glands are always and the sternoid mastoid glands sometimes involved, and exhibit the usual features of pri-

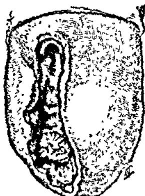


Fig 874.—Extensive gummatous ulceration of the tongue (*Musée de Saint-Louis*).

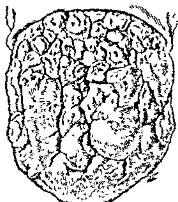


Fig 875.—Tertiary syphilitic sclerosis of the tongue (*Musée de Saint-Louis*).

mary syphilitic glandular enlargements (one large node with surrounding smaller nodes, practically painless; see *Neck, glandular enlargements in*). The history of infectious contact, the examination for the spirochete, and eventually the appearance of the roseola constitute, as always, the chief diagnostic factors.

**Secondary lingual syphilides** (mucous patches) are usually discrete and demand a careful search. They occur in the form of round or elliptical patches, of smooth "depapillated" appearance, contrasting with the normal granular background of the lingual mucous membrane.

**Tertiary syphilides** occur in two main clinical forms, *viz*., the *sclerous* and the *gummatous*, which in exceptional instances are combined in a mixed *sclero-gummatous* form.

Gumma of the tongue may be superficial or deep. When *superficial*, it causes local elevations of the dorsum in the form of multiple small nodosities varying in size from a leaden shot to a pea, and sometimes arranged in a horse-shoe-shaped figure. If untreated, it softens and ulcerates, leaving a relatively deep, cyclic or polycyclic ulcer with clear-cut margins. When *deep*, it is situated in the midst of the muscles of the tongue, forming there a hard node, nearly spheric or ovoid in shape, of hazelnut to walnut size, painless, and unaccompanied by any glandular reaction If allowed to progress unopposed, it enlarges and involves the dorsal surface of the tongue, softens, and ulcerates, discharging a characteristic gummy fluid This constitutes the deep *tertiary syphilitic ulcer*, with punched out margins, discharging base, not bleeding, and unattended with any glandular reaction unless infection or secondary degeneration sets in.

Fibrous glossitis, like gumma, may be either superficial or deep-seated.

When superficial, the cords and patches of fibrous tissue form irregular islets of superficial induration, sometimes smooth, at other times leucoplastic, the tongue at times assuming a stringy appearance owing to the presence of a network of shallow fissures lined with fibrous tissue.

When deep-seated, they infiltrate the major portion of the tongue, especially its anterior region, imparting to it a hard, wood-like consistency The simultaneous presence of large and small knob-like elevations, lobulated, smooth, and devoid of papillæ, gives to the tongue an absolutely pathognomonic aspect (lingual cirrhosis). Like gummatous glossitis, fibrous glossitis is not accompanied by any secondary glandular enlargement and is practically painless These two features should never be overlooked, as they are of capital diagnostic import.

**III. A Tumor-like Enlargement is Present**.—In most instances the patient first notices that certain movements of the tongue are somewhat hard to execute Clinical examination thereupon reveals some abnormality in the size and consistency of the organ.

Brief reference may be made, as an *altogether exceptional* possibility, to cysts (either parasitic, glandular, or congenital), which

are very hard to diagnose. Unless an exploratory puncture is made, there can be *nothing more than a mere presumption* of the presence of a cyst, based on its fluid consistency—which is, however, not easy to detect—and on the history. The same is true of the *connective tissue tumors, viz.*, lipoma, fibroma, fibrosarcoma, and chondroma.

In the presence of a tumor-like enlargement of the tongue, the chief possibilities which should come to the physician's mind are syphiloma, epithelioma, tuberculoma, and actinomycosis.

Nothing more need be said concerning the syphilomas, the customary features of which have already been briefly referred to —gumma and fibrous glossitis, characterized by painlessness and the absence of glandular enlargement.

The condition which has most often to be differentiated is **epithelioma.** In theory such differentiation is easy. The epithelial induration present shows a special consistency; it is imperfectly circumscribed, having a tendency to infiltrate the neighboring tissues, it is attended with a varying degree of tenderness, or may cause actual pain; the accompanying glandular enlargement consists at first of small separate nodes, which roll beneath the finger and coalesce only at a late stage of the disorder; finally, the condition occurs only in elderly subjects and its tendency toward progressive extension to surrounding tissues is manifest and obstinate Nevertheless, the actual distinction of certain hypertrophic forms of epithelioma of the tongue from fibrous glossitis is sometimes a matter of great difficulty and baffles even the most astute clinicians, particularly in view of the fact that there occur mixed forms giving rise to a hybrid cancerosyphilitic glossitis—a condition which has been carefully studied by Verneuil. The physician is thus easily induced to institute the therapeutic test by brief but active administration of potassium iodide and mercury or potassium iodide and arsenicals—a procedure which rapidly dissipates the disease if it is syphilitic or, on the other hand, stimulates it temporarily to greater activity if epithelioma is present

**Tuberculomas,** which are very uncommon in the tongue as compared to the syphilomas and epitheliomas, appear usually on the upper surface of the organ They are sensitive or painful on pressure, engorgement of lymph-nodes is exceptional, the

patient is always a tuberculous individual, and, if necessary, excision of a bit of the tumor tissue for examination and injection of the diseased tissue into a guinea-pig will lead to recognition of the presence of the tubercle bacillus

**Lingual actinomycosis** may so resemble fibrogummatous syphilis or a deep tuberculoma as to be readily mistaken for it If the condition is only thought of, however, the diagnosis may be settled by exploratory puncture and examination on a slide (with cover-slip) of the characteristic small yellow granules, sug-



Fig 876—Actinomycosis

gesting powdered iodoform, which the fluid withdrawn allows to settle on the walls of the tube into which it has been expressed When stained with picrocarmin they appear as if made up of a central felt-like central mass consisting of mycelial filaments and surrounded by a radiating zone of ovoid bodies disposed like petals, the whole resembling to some extent a daisy.

Lastly, mention may be made of a congenital anomaly, fortunately very rare, constituting a real pathologic curiosity, *viz.*, macroglossia, which, undergoing development during later childhood and even the period of puberty, may lead to permanent prolapse of the tongue, which has become too large for the buccal cavity.

**IV. Ulcerations are Present.**—Small ulcers on the tongue may be observed:

1. During an attack of whooping-cough; there is ulceration of the lower surface of the tongue and its frenum, due to *forcible* projection of the tongue against the teeth during cough.

2. As a result of biting of the tongue; there is *present* a cut-like ulceration along the margins of the organ, *e.g.*, in *epilepsy*.

3. As a result of dental traumatism, the ulcer being situated opposite a badly decayed, broken, or deviated tooth.



Fig 877—Macroglossia (*Mikulicz* and *Kümmel*).

The more extensive ulcerations of the tongue are due to the four following major causes of tumor formation.

| | |
|---|---|
| *Syphilitic ulcers.* | *Tuberculous ulcers.* |
| *Neoplastic ulcers* | *Actinomycotic ulcers.* |

**Syphilitic ulcers** are represented mainly by ulcerated gummas; mention need here be made only of their characteristic depth, perpendicular margins, sloughing base, absence of bleeding, and relative painlessness, as well as the usual absence of glandular enlargements and the history. In the event of doubt the therapeutic test will settle the diagnosis

**Neoplastic ulcers**, or epitheliomatous ulcers, show lacerated, irregular margins, with suppurating bases, and oozing of blood, and are superimposed on an indurated mass which infiltrates the

neighboring tissues There is copious salivation and a malodorous breath. Such ulcers give rise to pain, sometimes of marked severity, radiating toward the ears. The submaxillary lymphatic ganglia are involved.

**Tuberculous ulcers** are generally broad and superficial, multiple, with clear-cut, but only slightly elevated margins, with a

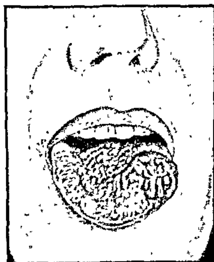


Fig 878—Tuberculosis of the tongue

pale, grayish base, not prone to bleed, but causing much pain. Sometimes they are surrounded by discrete yellowish points or granulations resembling millet seeds, constituting actual small tuberculomas, which would be pathognomonic were similar manifestations not sometimes observed in actinomycosis

**Actinomycotic ulcers** may present an appearance similar to that of the preceding type of ulcers, they exude serous pus, in which the characteristic yellow granules already referred to should be sought.

## TREMOR. [Tremulare, *to tremble.*]

---

Tremor consists of regular oscillatory movements taking place at a varying rate and with varying amplitude, involving the entire body or an extremity, and symmetric in both directions from the resting position. The mode of production of tremor is still obscure and a mooted subject.

Tremor may be either accidental (transitory) or essential (permanent).

I. **Accidental (transitory) tremor** is chiefly:

(*a*) **Emotional,** in which it is easily traced to its cause, *e g*, fear or other emotional stress

(*b*) **Pyrexial,** frequently marking the onset of an acute infection (see *Chills*)

The initial chill of pneumonia and that occurring in the first stage of the malarial paroxysm are well-known examples, both of which are as a rule traceable to their cause.

(*c*) **Cryogenic,** or brought on by cold, and in the initial stage of fevers "You are trembling, Bailly?—Yes, but it's because I'm cold!"

II. **Permanent tremor** occurs in two forms·

Tremor during the execution of volitional movements (kinetic or intention tremor), typically exemplified in *disseminated sclerosis*

Tremor at rest (static tremor), exemplified in *paralysis agitans*

A. **Tremor during voluntary motion.**—Charcot ascribed this form of tremor to the usual persistence of the axis cylinder deprived of its myelin sheath in the midst of the areas of sclerosis Pierre Marie compares such an axis cylinder to an electric wire from which the insulation has been removed and along which an escape of electricity occurs causing the tremor

(*a*) This type of tremor is illustrated in the highest degree and in the utmost state of purity in *disseminated sclerosis.* While

absent during rest, it starts up when movements are undertaken. It quickens, develops, and exhibits increased amplitude during the execution or repetition of the motor act. The rate of vibration is always moderate, *viz.*, six or seven vibrations per second The amplitude, which increases during the performance of the motor act, may become very great—5, 10, or even 30 or 40 centimeters.

In this disease the tremor is accompanied by vertigo or apoplectiform seizures, by ocular disturbances and nystagmus, by exaggerated reflexes, and sometimes by spastic paralysis and slow, scanning speech.

(*b*) *Hereditary tremor* (with a similar tremor or merely a neurotic tendency in the parents) may be considered to be present when the tremor is absent during complete rest, exhibits a rapid rate of vibration, develops with movements but does not increase during their execution, involves particularly the upper extremities, eyelids, lips, and face, and set in slowly during childhood or adult life. It is sometimes present to a slight extent during rest.

(*c*) *Toxic tremor,* due to alcohol, coffee, tea, lead, mercury, etc, is remittent and of the intentional type, of intermediate rate, and but slightly marked or absent during rest A brief inquiry as to the subject's past history will generally settle the diagnosis. This is by far the commonest kind of tremor

(*d*) Tremor is said to have been observed sometimes in the secondary stage of *malaria.*

B Tremor during rest (static tremor) is but slightly or not at all affected by voluntary movements.

(*a*) This variety of tremor is most characteristically exemplified in paralysis agitans (Parkinson's disease) In contrast with the preceding variety, it is continuously present during rest and ceases—at least in the earlier stage of the disease—during the performance of voluntary movements It is a slow tremor, exhibiting four or five vibrations per second, of slight amplitude, and coordinated, simulating the endless repetition of voluntary movements—pill rolling, thread winding, crumbling of bread, masturbation, etc.

It involves chiefly the upper extremities, the head and neck being relatively free It is accompanied by a special kind of muscular rigidity (waxy flexibility) and a slowness in starting

locomotion which impart to the act of walking a very typical slow and scanning appearance. There are also noted a subjective sensation of heat and a morbid desire for locomotion.

Grasset called attention to two anatomic regions often found the seat of morbid changes in this disease:

1. The region of peri-ependymal myelitis, with obliteration of the central canal

2. The automatic pontobulbar centers and the capsulothalamic region

(*b*) The commonest form in this class of tremors is perhaps **senile tremor,** which, as the term implies, occurs particularly in old persons, is of comparatively slow rate and, affecting mainly the head and neck, imparts to the tremor the peculiar suggestion of an indefinitely repeated nod of affirmation or shake of negation

(*c*) **Graves's (Basedow's) disease.**—Here the tremor shows a rapid rate. It generally occurs in conjunction with the symptomatic triad, tachycardia, goiter, and exophthalmos. This triad may, however, be absent or only partially represented, in which *event one should look for the other signs of hyperthyroidia*—frequent pulse, tremor, hyperidrosis, exaggerated reflexes, and diarrhea.

(*d*) **Tumors of the cerebral peduncle.**—In tumors of the crus, the tremor presents the same features as in Parkinson's disease, but is generally unilateral.

(*e*) **Post-hemiplegic tremor** is sufficiently characterized by the history of hemiplegia and exaggeration of the reflexes.

(*f*) The **tremor of progressive general paralysis** exhibits a rapid rate; it is accompanied by a special difficulty of speech, by pupillary disturbances (Argyll-Robertson pupil), and by special, characteristic changes of mentality.

(*g*) Meige has called attention to the occurrence of tremor of the type of paralysis agitans in wound cases.

III. **Hysteric Tremor.**—A special division is required for *hysteric tremors.* These do not belong in any of the foregoing categories. Their various modalities closely imitate most of the other forms of tremor. How are they to be differentiated, then, from such tremors,

and in particular, from toxic, exophthalmic, Parkinsonian tremors, etc.? Such differentiation is often rendered all the more difficult in that hysteria is frequently combined with most varied forms of intoxication and neurosis.

On the whole, the diagnosis of hysteric tremor is founded on two groups of symptomatic features:

1. *Positive* indications: Sudden onset, following convulsive seizures, in a subject exhibiting sensory and special sense stigmata of hysteria; possibility of change in the type of tremor present; curative influence of suggestion

2. *Negative* indications. Absence of the usual signs of hyperthyroidia, of disseminated sclerosis, of exaggeration of the reflexes, etc.

* * *

The **clinical course** exhibited by the tremor may also be of diagnostic assistance:

1 *Progressive tremor*, at first localized in one limb or portion of a limb, progressively extends to one or more other limbs Such is the case in the tremor of paralysis agitans

2. *Retrogressive tremor*, generalized at the start, tends gradually to become localized in one limb or portion of a limb This kind of a clinical course obtains particularly in tremor of neuropathic origin.

3 *Migratory tremor*, exhibiting variable localizations both as regards space and time. Such changes in the situation of the tremor may come on spontaneously; they are rendered more distinct by changing the positions of the limbs or immobilizing one or more portions of the limbs This is frequently the case in hysteric tremor.

* * *

The **relative order of frequency** of the various types of tremor as encountered in practice appears to be as follows:

1. Toxic tremor (alcohol, tea, and coffee).
2. Tremor of Graves's disease.
3. Neurotic, hysteric tremor
4. Senile tremor
5. Hereditary tremor
6. Tremor of paralysis agitans
7. Sclerotic tremor (disseminated sclerosis).

## TREMOR.

### I.—Accidental.

*Readily referred to its cause:*

(*a*) **Emotional:** Fear, pleasure, or other pronounced emotion.

(*b*) **Febrile:** (See *Chills.*)

(*c*) **A frigore:** Shivering.

### II.—Permanent.

**A. During motion** (as typified in **Disseminated sclerosis**).

| | |
|---|---|
| (*a*) **Disseminated sclerosis** | 1. Tremor absent during rest; sets in and increases with voluntary motion<br>2 Dizziness, apoplectiform attacks, nystagmus, exaggerated reflexes, and scanning speech |
| (*b*) **Hereditary tremor.** | 1 *Absent or slight during rest; sets in, without accentuation, during voluntary motion.*<br>2 None of the above associated signs; often sets in in childhood. |
| (*c*) **Toxic tremor** (tea, coffee, alcohol, etc) (by far the most frequent form) | 1. *Less extensive, but more rapid tremor than in the preceding disorders*<br>2 History of toxic poisoning |

**B. During rest** (as typified in **Paralysis agitans**).

| | |
|---|---|
| (*a*) **Paralysis agitans.** | 1 Persistent during rest; lessens or disappears *during voluntary motion*<br>2. *Special kinds of muscular rigidity; slow,* jerky locomotion, characteristic facies and posture |
| (*b*) **Graves's disease.** | Tremor associated with frequent pulse, goiter, and exophthalmos |
| (*c*) **Post-hemiplegic tremor.** | History of hemiplegia; exaggerated reflexes |
| (*d*) **Progressive general paralysis.** | Special speech disorder; pupillary disturbances (Argyll-Robertson pupil); special and characteristic mental changes, history of syphilis |

### III.—Hysteric.

(*a*) Abrupt onset after a convulsive seizure; sensory and special sense stigmata; variability of the type of tremor.

(*b*) Absence of concrete somatic evidences

(*c*) Mythomania.

# UPPER EXTREMITIES, PAIN IN.

This section on pains in the upper extremities is undertaken by the author with some degree of trepidation, as such pains have seemingly little to do with "internal medicine." Yet the author has frequently found himself embarrassed in the interpretation of some of these pains While often of obvious causation, as in herpes zoster or acute articular rheumatism, they are at times

In the succeeding illustrations, borrowed from the writings of Mme Benisty, and showing diagrammatically the innervation of the upper extremity, the fields of distributions of the different nerves are to be recognized by the kind of shading, as follows.

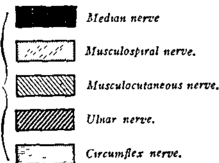



Fig 879

very obscure, as in many vasomotor disturbances concerning which little is as yet known

In such cases, it is necessary to carry out a careful, systematic anatomo-clinical analysis, which, while indispensable, unfortunately does not always yield clear-cut information

Any of the tissues forming part of the upper limb may be the source of painful disturbances; many brachialgias constitute the outward manifestations of some spinal or deep visceral disorder, ordinarily mediastinal and in the majority of instances aortic or peri-aortic.

Aside from the *post-traumatic affections* (fractures, luxations, sprains, wounds, and contusions), the diagnosis of which is generally obvious, the joint disorders greatly predominate as causes of pain in the upper extremity, whether localized in the shoulder,

elbow, the joints of the carpus or metacarpus, or the phalanges. Reference need not here be made to all the possible causes of *articular pains*, a special section having been devoted to the subject (see *Joint pains*). Systematic examination by inspection, palpation,

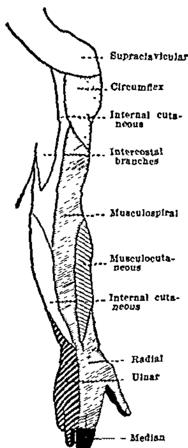



Fig. 880—Peripheral sensory distribution in the upper extremity (posterior aspect).

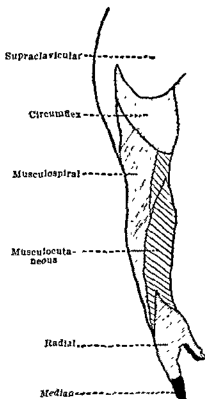
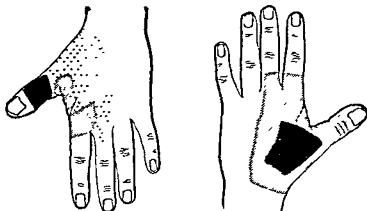



Fig 881—Peripheral sensory distribution in the upper extremity (lateral aspect).

and passive motion will first of all localize the pain in one of the above mentioned joints. The special features of the joint disturbance, the history, the mode of onset, and the subsequent course will, as a rule, enable the observer quickly to class the condition under one of the following heads, *viz*, acute articular rheumatism, gonorrheal rheumatism, tuberculous rheumatism, post-infectious (*e.g*, post-typhoid) rheumatism, gout, arthritis deformans, etc. One can-

not too earnestly recommend to the practitioner, as in other disorders, to examine the painful region carefully and by actual inspection, to palpate and mobilize it; in short, to localize with care the pain and the actual local condition present, to determine its nature if possible, and especially, not to rest content with the indefinite term "rheumatism,"—a term just as devoid of true diagnostic meaning as "headache" or "pain in the side."

The examiner should not forget the possibility of *tabetic joint disease*, which, while an exceptional disorder, presents characteristic



Figs 882 and 883—Two common types of distribution of the sensory disturbances in complete section of the radial nerve Black area: Anesthesia to all stimuli except deep pricking, which is often felt as a mere contact with the tissues Gray area Marked hypoesthesia to pricking, anesthesia to heat, cold, and very superficial tactile stimuli. Dotted area Slight hypoesthesia to tactile stimuli and to heat and cold. (*Mme. Benisty*)

features consisting of a striking degree of deformity, extreme relaxation of the joint, and painlessness. If only the condition is kept in mind, the diagnosis can be made by observation of the other signs of tabes, [illegible], specific history, reflex disturbances (Argyll-Robertson pupils and loss of knee-jerks), astasia, ataxia, lightning pains, sphincter disturbances, etc.

Disorders of bones—osteitis, osteoperiostitis, osteoarthritis, and osteomyelitis—are, in the order of their frequency:

*Tuberculous* Periostitis and osteoarthritis (white swelling).

*Syphilitic:* Osteoperiostitis and gumma.

elbow, the joints of the carpus or metacarpus, or the phalanges. Reference need not here be made to all the possible causes of *articular pains*, a special section having been devoted to the subject (see *Joint pains*). Systematic examination by inspection, palpation,

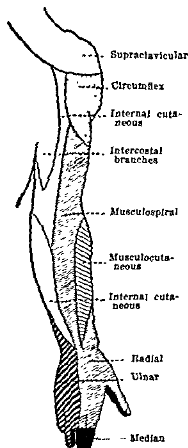



Fig. 880.—Peripheral sensory distribution in the upper extremity (posterior aspect).

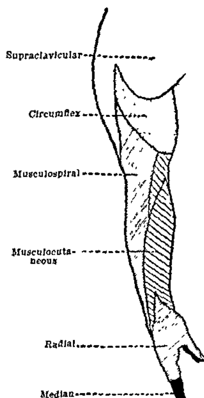



Fig. 881.—Peripheral sensory distribution in the upper extremity (lateral aspect).

and passive motion will first of all localize the pain in one of the above mentioned joints. The special features of the joint disturbance, the history, the mode of onset, and the subsequent course will, as a rule, enable the observer quickly to class the condition under one of the following heads, *viz.*, acute articular rheumatism, gonorrheal rheumatism, tuberculous rheumatism, post-infectious (*e.g.*, post-typhoid) rheumatism, gout, arthritis deformans, etc. One can-

6. Special tests such as the Wassermann reaction and the successful therapeutic test (mercury and iodide) in syphilis

Disorders of the muscles and serous surfaces, viz., *myositis, subacromial bursitis,* and *deltoid rheumatism,* are possible localizations of rather poorly defined painful manifestations, the origin of which, however, seems actually to be that mentioned by Le Gendre: "Deficient functioning of the locomotor apparatus, either through lack of exercise (sedentary mode of life) or through excessive muscular labor." Such a deficiency through loss of functional balance,

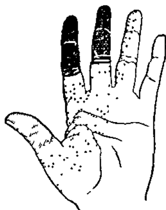


Fig 885—Palm of the hand

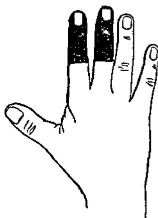


Fig 886—Dorsum of the hand

*Distribution of the disturbances of sensation in severe injuries of* the median nerve Black area Complete anesthesia of all types Gray area Hypoesthesia to pricking and anesthesia to heat and cold Dotted area Less marked hypoesthesia.

"when it has been present in patients, necessarily places the various component parts of the locomotor apparatus in a weakened state in which it becomes susceptible to influences ordinarily not noticed by well persons; these are the influences to which we are constantly exposed, *viz.*, the *cosmic influences.* Among these influences, the best studied has been that of cold, which is even considered an etiologic factor—especially damp cold and long-continued cold or exposure"

The upper extremity may be, and manifestly is, the seat of neuralgia and neuritis variously situated (*e.g*, ulnar, radial, or median) and of varying cause: Traumatic (contusions, open sur-

*Post-infectious:* Staphylococcic (osteomyelitis).

*Neoplastic:* Osteosarcoma.

The following features will insure a proper diagnosis:

1. The history: Lymphatic diathesis, specific taint, infection (typhoid fever or staphylococcic infection).

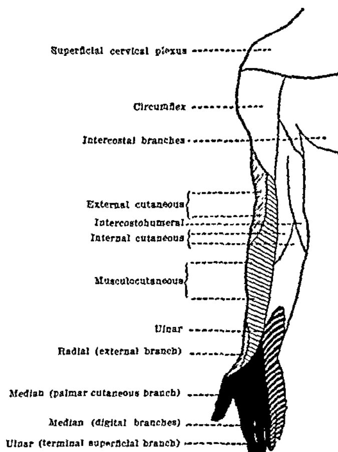



*Fig 884.—Peripheral sensory distribution in the upper extremity (anterior aspect).*

2. Fever (generally wanting in syphilis and neoplasms).

3. Localization particularly around the joint in tuberculous disease.

4. A rather sluggish clinical course in tuberculosis and syphilis.

5. The nature of the pain, with nocturnal exacerbations in syphilis.

left dorsal nerve over the chest and arm. It may descend and radiate as far as the epigastrium in the areas of distribution of the fifth and sixth dorsal nerves. More often, however, it ascends and radiates along the first, second, and third dorsal or even the seventh and eighth cervical nerves, being felt, therefore, along the ulnar

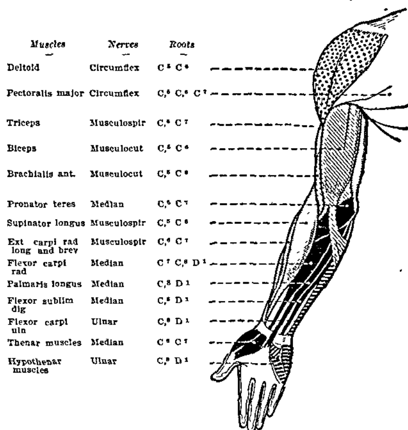



Fig 887—Nerve-supply of the muscles of the upper extremity (anterior aspect)

border of the forearm and hand This constitutes the typical area of distribution of anginose pain, *viz.*, precordial (mammillary), left upper thoracic (axillary), and ulnar

Exceptionally, the pain extends to the corresponding areas on the right side (chiefly in cases with aortic dilatation) and to the neck, including its posterior aspect (nuchal region).

faces and wounds). toxic (lead poisoning, alcohol, etc.), pressure (osteoperiostitis, osteosarcoma, or defective callus of the humerus, radial paralysis of lovers, etc.), and infections. Careful examination for local sensitiveness (see illustration showing the distribution of the sensory nerves), as well as of mobility (paralysis; see illustration showing distribution of the motor nerve fibers) will soon lead to discovery of the site of the lesion. The history and even the location of the neuritis will frequently permit of tracing the cause of the disturbance

Lastly, mention may be made of vasomotor disturbances, represented in the highest degree by *Raynaud's disease* (local asphyxia of the extremities), and seemingly often of specific origin, whether acquired, inherited, or secondary to a mitral malformation.

Exceptionally, in particular among the Senegalese, there may be noted *evidences of leprosy* of the nervous or anesthetic types, the *nerve symptoms being manifest in a prodromal stage by a moniliform* enlargement, frequently of considerable size, of the nerve trunks, which are very sensitive to pressure; later, the pain, of neuralgic type, is constant and spontaneous, with repeated violent paroxysms; it is accompanied by dysesthesias (itching, etc.) and by vasomotor disturbances ("dead finger" sign), and ends in anesthesia, with trophic disorders, atrophy of muscles, and deformity. These manifestations of leprosy exhibit great clinical analogy with *syringomyelia* and with *"Morvan's disease"* or panaritium analgicum

**Pain in the arm occurring in the absence of any local lesion,** whether muscular, articular, osseous, nervous, or vascular, *may constitute the outward expression of remote lesions,* the most important of which are:

(*a*) *Aortic and periaortic lesions* (radiation of the pains of angina pectoris and aneurysm to the arm).

(*b*) Certain "high" forms of degeneration of the posterior columns (lightning pains of *cervical tabes*).

(*c*) *Lesions of the brachial plexus.*

(*d*) Certain *mediastinal tumors.*

(*a*) The *pain of angina* and of aortic and periaortic disorders in general is, as a rule, felt in the area of distribution of the fourth

venous network in the superior caval area, swelling of the neck, edema of the face, and inequality of the pulse on the two sides, etc.), are obvious; fluoroscopic examination will remove all doubt as to the condition present

(*c*) The same is true in *tumors of the mediastinum.* The coexistence of pain in one or both upper extremities with evidences of interference with the return circulation in the superior caval area (cyanosis, visible superficial venous circulation, swelling of the neck and face, prominence of the eyeballs, and facial edema) is almost pathognomonic of mediastinal tumor, *e g*, dilatation of the aorta, pericarditis with effusion, enlarged lymphatic glands, etc.

(*d*) The *pains of tabes,* although much less common in the upper than in the lower extremities—the latter constituting the site of election—may occur as the usual "lightning pains," sharp, abrupt, and fulgurant, like lightning Sometimes they occur singly, at others grouped in paroxysmal attacks of varying duration—from one to several days They are felt more particularly along the inner border of the forearm and the fifth and fourth fingers. They may be of the piercing, burning, lancinating type, at times, however, they are of the nature of a constriction, squeezing, or ring-like pain. This special characteristic of the pains of tabes—though not an exclusive attribute, since it may be met with likewise in peripheral neuritis (due, *e.g*, to alcoholism or leprosy) and in pressure on nerve-roots—is nevertheless, as a rule, highly suggestive. Let the observer merely recollect the possibility of tabes—and how could he fail to think of it in such cases?—and the diagnosis will be quickly confirmed by examination for the typical tabetic indications (specific history, reflex disturbances, motor disturbances, visceral disturbances, particularly genitourinary, etc.).

* * *

The foregoing review by no means exhausts the possibilities as regards pain in the upper limbs. Various exceptional clinical conditions, such as *supernumerary cervical rib, poliomyelitis,* etc., have been designedly omitted in order not to make the present section too unwieldy.

The same applies to *abscesses, felons, lymphangitis, phlegmonous inflammations,* and the attendant *glandular enlargements*—all con-

Such pain is, as is well known, when present in conjunction with a feeling of constriction of the chest (squeezing sensation or suffocation) and a fear of impending death, the cardinal indication of *angina pectoris.* It should always be borne in mind, however, that many neuropathic states, whether or not based on some cardioaortic disease, may lead to a syndrome of "anxiety neurosis" similar in all respects to the anginose syndrome, yet far more favorable from the standpoint of prognosis. For a discussion of the differential diag-

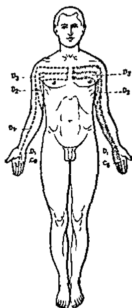


Fig 888—Distribution of the pain and cutaneous hyperalgesia after repeated attacks of angina pectoris

nosis in this connection the reader is referred to the section on *Precordial pain.*

(*b*) *Aortic aneurysm* may cause pain in the arm of threefold origin:

1. Anginose pain of the type above described.
2. Neuralgic or neuritic pressure pain.
3. Pain due to stasis on account of pressure on venous trunks.

These pains may be of unbearable intensity and necessitate the use of morphine. Often the clinical evidences, such as aneurysmal swelling and signs of pressure on the veins (visible

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**Vertigo** is characterized by a mistaken subjective sensation in virtue of which the patient believes himself to be rotating although he is motionless, or sees surrounding objects turning about him although they are at rest When vertigo is persistent or very marked, it may induce loss of equilibrium and a fall to the ground if the subject is in the standing position at the time. It may be, and frequently is, attended with nausea, or even vomiting, and by nystagmus or even deviation of the eyes.

The highly complex pathogenesis of this symptom seems, at the present time, to be rather well condensed in the following definition formulated by Grasset: *Vertigo is the symptom of functional insufficiency (intermittent claudication) of the automatic centers (mesencephalic and cerebellar) of equilibration.* Bonnier, as is well known, has made a profound study of vertigo from the physiopathologic, clinical, and therapeutic standpoints.

**These automatic mesencephalic and cerebellar centers of equilibration:**

1. *Receive:*

(*a*) Vestibular fibers coming from the semicircular canals through the auditory nerve

(*b*) Visual fibers coming from the retina through the optic nerve.

(*c*) Muscle-sense fibers from Clarke's columns and the posterior columns

2. *Send*

(*a*) Fibers terminating in the Rolandic area on the opposite side and acting on the motor centers

(*b*) Fibers terminating in the nucleus of Deiters, which is connected with the oculomotor nerves, these in turn governing the ocular muscles.

(*c*) Fibers terminating in the anterior horns of the spinal cord, whence radiate motor fibers to the voluntary muscles.

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(*d*) Fibers to other bulbar centers (glossopharyngeal and pneumogastric).

*These anatomic and physiologic connections of the mesencephalic and cerebellar centers account for the following:*

(*a*) The clinical combination of vertigo, disturbances of equilibrium, nausea, and ocular disturbances (nystagmus and deviation of the eyeballs). (Normal equilibrium is the result of harmony of the retinal, labyrinthine, and muscular impressions).

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1. Vestibular fibers
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3 Acoustic nuclei of Bechterew and Deiters
4 Visual fibers
5 Corpora quadrigemina.
6. Sensory fibers
7 Columns of Clarke
8. Cerebello-rolandic commissural fibers
9 Cerebello-auditory fibers.
10 Cerebello-oculomotor fibers.
11 Cerebello-spinal fibers
12. Cerebello-bulbar fibers.
13 Muscles.
14. Retina

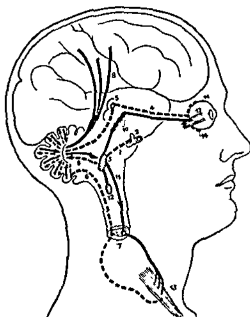


Fig 889—Pathogenesis of vertigo (*Bonnier*). The cerebellum appears as the "vertigo center," whence the frequency, if not the uniform presence, of vertigo in cerebellar disorders

(*b*) The factors generally causative in vertigo:

1. *Disorders and pathologic changes in the centers of equilibration* (cerebellum and crura).

2. Wrong or disharmonious impressions supplied from the receptive structures (retina, labyrinth, and muscle-sense).

3 Morbid stimuli in the field of the pneumogastric nerve (gastric disorders, helminthiasis, etc).

From the foregoing brief and synoptic enumeration the reader will readily conceive how common vertigo may be and

in how many different varieties it may occur, and what powers of clinical analysis will frequently have to be brought to bear in seeking its causes (auricular vertigo, arteriosclerotic vertigo, cerebral vertigo, neurotic vertigo, migrainous vertigo, ocular vertigo, epileptic vertigo, toxic vertigo, etc.)

Under physiologic conditions, vertigo may be brought on by abrupt change in the position of the body, *e.g.*, rising from recumbency to the standing posture, by an abrupt change of direction or motion, by rapid rotation, by an ascent, and by looking down from a high place. Travelling in a railway or trolley car suffices, as is well known, to induce vertigo in some persons Vertigo is an essential component of seasickness. The electric current, in particular the application of the galvanic current to the head, or sharp percussion of the middle ear by use of the aural syringe, will induce vertigo in predisposed individuals

Clinically, one cannot here do more than recall in a concise table the more usual causes of vertigo, which investigation of the concomitant symptoms will alone identify:

## I.—CENTRAL VERTIGO.

*Central vertigo occurs mainly when the position of the body is changed,* e.g., *upon passage from the horizontal to the vertical position.*

**Arteriosclerosis.**—The highest degree of importance should be attached to vertigo coming on without apparent cause in elderly individuals previously never afflicted with it. Old age, vertigo and high blood-pressure, present in combination, nearly always mean arteriosclerosis The vertigo may be of mild degree, even in this type of case, and remain so for years; sometimes—too often—it is an alarm signal betokening an approaching and threatening attack of apoplexy.

**Cerebral tumor.**—Vertigo is observed in the majority of cases of frontal tumor and in over one-third of cases of tumor in other regions of the cerebrum; the attacks of vertigo seem to correspond to the periods of enlargement of the tumor. Examination of the eyegrounds for choked disc should never be neglected

(*d*) Fibers to other bulbar centers (glossopharyngeal and pneumogastric).

*These anatomic and physiologic connections of the mesencephalic and cerebellar centers account for the following*·

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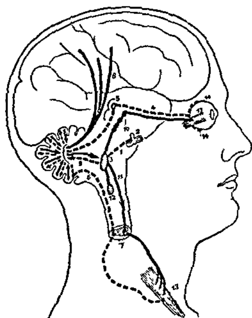


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From the foregoing brief and synoptic enumeration the reader will readily conceive how common vertigo may be and

hemorrhage into the internal ear.[1] Thus, only a very small number of cases served in the earliest description of this disease

Since then, the meaning of the term has been expanded, and whenever a patient presents aural vertigo he is said to be suffering from Ménière's disease. This is a mistake, however, for while the symptom is the same, the underlying pathologic condition is entirely different, and it is well to bear in mind that any disease of the *ear* (external, middle, internal, Eustachian tube, tympanic membrane, and tympanic cavity, . . . *e.g.*, an ordinary impaction of earwax, or suppurative otitis) is capable of bringing on vertigo with deafness and tinnitus This occurs through irritation of the labyrinth. Thus, given a plug of wax in contact with the tympanic membrane and forcing in the latter, and consequently also the chain of ossicles, irritation of the labyrinth results; and the same is true when there is increased tension of the labyrinthine fluid from the presence of pus in otitis, and where there is anemia, congestion, or infection of the internal ear in certain constitutional disorders or through poisoning by drugs such as quinine and sodium salicylate.

To recapitulate

1. The term *Ménière's disease* should be exclusively set apart for vertigo induced by an actual hemorrhage into the labyrinth. This condition is very uncommon

[1] Ménière's initial case is deserving of reproduction; he studied it in Chomel's service, being at that time on the staff of the Hôtel-Dieu.

"A young girl, having travelled at night on the top of a stage-coach during her menstrual period, was seized, after considerable exposure to cold, with sudden and complete deafness Upon admission in Chomel"s service she showed constant vertigo as the chief symptom the least attempt to move brought on vomiting, and death took place on the fifth day

"The necropsy showed the cerebrum, cerebellum, and spinal cord to be completely free of any pathologic change, but inasmuch as the patient had become suddenly deaf after having always enjoyed good hearing, I removed the temporal bones in order to make a careful inquiry into what might be the cause of this complete deafness of such sudden advent. I found nothing in the way of a pathologic change save the filling of the semicircular canals with a plastic red material, a species of bloody exudation, of which only traces could be seen in the vestibule and which was entirely absent from the cochlea Careful investigation enabled me to establish with all necessary accuracy that the semicircular canals were the only structures exhibiting an abnormal condition in this case."

in suspected cases, and other signs of brain tumor, such as vomiting, etc., should be inquired for.

**Cerebellar tumor.**—Vertigo is almost constant in tumors of the cerebellum; in nearly all instances it is associated with disturbances of equilibrium which lead to a staggering gait, lateropulsion, a gait as of "pseudo-intoxication"—the cerebellar gait, which is highly significant to a practised eye.

**Disseminated sclerosis.**—Vertigo is one of the most frequent signs of disseminated sclerosis, being combined with nystagmus and the intention tremor characteristic of this disorder.

**Paretic dementia.**—Vertigo occurs in two stages of this incurable affection, *viz.*, at the start, in the preliminary stage, and in the advanced stage, as a precursor of some acute complication, such as coma, convulsions, or hemiplegia.

**Disturbed circulation in the centers,** whether of hyperemic or anemic type, and whether dependent upon actual congestion, actual anemia, or vasomotor disturbances, may be an exciting cause of vertigo. Such is the vertigo of syncope (in its premonitory stage), of the menopause, of chloroneurosis, of the anemias, of hemorrhage, of heart disorders, etc

## II.—AURAL VERTIGO.

### Ménière's vertigo —Labyrinthine vertigo.[1]

(By G Laurens, M D)

**What is meant by aural vertigo?**—One portion of the ear, as is well known, is concerned in *audition*, and the other in *equilibration, i e.*, in orientation of the head, which plays an important rôle in the maintenance of body balance. These two portions are independent:

Disease of the one causes *deafness* and *tinnitus.*

Disease of the other causes *vertigo.*

**What is Ménière's disease?**—About fifty years ago Ménière described a disorder characterized by the following *three symptoms: vertigo, deafness,* and *tinnitus,* and on post-mortem examination of the *few* cases upon which his contribution was based, observed

[1] From Laurens: *Loc. cit.*, p. 132 ff.

(*b*) In some instances, the subject is a person in good health in whom vertigo appears without the least apparent cause—and this is frequently the case;—in other instances he is already deaf, suffering from otosclerosis, or acute or chronic suppurative otitis; or, again, in the course of some general infectious disease, or sometimes following ingestion of drugs, such as quinine and salicylates, he was seized with the symptoms already mentioned, *viz.:*

**Vertigo,** causing staggering, failing to disappear in recumbency or upon closure of the eyes, drawing the patient toward the side of the affected ear, and lasting a few hours; sometimes inducing a *continuous* state of malaise with dizziness; acute **tinnitus,** with

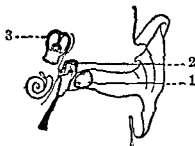



Fig 891—The causes of aural vertigo Aural vertigo may be the result of a disorder of the external ear (*1*) (foreign body, wax in contact with the drum, etc), of otitis media (*2*), of hemorrhage in the labyrinth (Meniere s disease), or of anemia, hyperemia, or suppuration of the labyrinth, intoxication by drugs, or general, infectious diseases. This is the common form of aural vertigo

loud whistling sounds; increasing **deafness.** This symptom-group is at times supplemented by bilious **vomiting,** appearing spontaneously, without the least exertion, and which the physician is led to ascribe to dyspepsia

Such is the *ordinary type of aural vertigo.* Its existence must be confirmed:

2. By Otoscopic Examination.

The observer finds in some instances a plug of wax, sometimes an otitis, and in still others an *absolutely normal tympanic membrane;* this last means that the vertigo is of labyrinthine origin.

2. There occurs also a *syndrome of Ménière,* or, more commonly, aural vertigo, when there is irritation of the internal ear from any cause.

**How is one to recognize the existence of aural vertigo?**

1. From the nature of the disturbances complained of by the patient.

2. By otoscopic examination

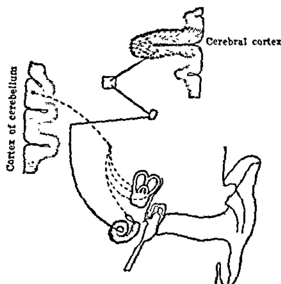



Fig 890—Diagram illustrating aural vertigo

The two portions of the internal ear. 1 The auditory portion, represented by the cochlea and related to the cochlear nerve 2. The organ of *equilibration* or of the spacial sense, represented by the vestibule and semicircular canals and related to the vestibular nerve

**1. From the Nature of the Disturbances Complained of by the Patient.**

(*a*) Sometimes he will tell you that while in perfect health, without any noticeable cause, he felt a sort of explosion or intense sound in the ear. At the same moment, there was *tinnitus, dizziness,* sometimes an actual fall to the floor, with or without *nausea* and *vomiting.* This attack lasted from a few minutes to a few hours; as soon as it ended, the patient noticed that he was *deaf.* This major attack sometimes recurs, but *in a much less pronounced form.*

**Such is a typical case of Ménière's disease.**

ment directed toward the alimentary tract, *viz.*, diet, purgation, and stomach washing.

It is a fact that vertigo and vomiting or nausea, present in combination, may be obviously dependent upon a single cause which brings them on at the same time and is associated with and regulates them, as in seasickness, mountain sickness, car sickness, and, as we have already seen, brain tumors.

## VI.—VERTIGO WHICH RESTORES THE SENSE OF HEARING.

In a well-grounded and highly suggestive article entitled "Vertigo which restores the sense of hearing,—labyrinthine angiospasm," Lermoyez[1] described a curious vertiginous symptom-group characterized by abrupt and violent onset with recovery within a few hours of hearing in subjects whose auditory function had *previously seemed* hopelessly lost

"It appears to me," he wrote, "that one has to deal in these cases with *local angiospasms* in neuroarthritic (or, as I would rather put it, *gouty*) subjects possessed of exaggerated sensitiveness of the *internal ear, both as regards external stimuli (clattering sounds,* violent and prolonged movements) and internal irritants (various intoxications and especially gastric autointoxications)—which excuses Trousseau for having conceived his celebrated vertigo *a stomacho laeso!*"

Lermoyez compares the condition to the local asphyxia of the extremities with the characteristic painful numbness (*onglée*) which precedes restoration of circulation through the tissues

"Similar phenomena occur in the ears, and it would not be an exaggeration to speak of a 'painful numbness of the labyrinth.' The closure of the internal auditory artery, which took place slowly, induced gradual anesthesia of the ear, *i.e.*, deafness. But now the spasm abruptly relaxes; the blood rushes anew into the labyrinth, which it stuns, and this excessively sudden influx, which, in the fingers, causes the well-known pain, here induces simultaneously the special pain of the cochlear organ, which is tinnitus, and the suffering of the vestibular organ, which is vertigo; in addition, it causes disappearance of the anesthesia of the ear, *i.e.*, of the deafness."

[1] *Presse méd.*, Jan. 2, 1919

## III.—NEUROPATHIC VERTIGO.

1. In a first group should be placed **neurasthenia, psychoneuroses**, and the neuroses of congenital origin, in which there seems to be operative a congenital or acquired *hypersensitiveness* to stimuli and impressions of all kinds, sometimes with an added autosuggestibility which, for example, may automatically start an attack of vertigo in the presence of some factor—such as an odor, railway travel, riding in a carriage, etc.—which had already been previously associated with vertigo.

2. In a second group we shall place **epilepsy**, in which vertigo may be encountered either as a premonitory aura or as an equivalent of the epileptic seizure.

## IV.—TOXIC VERTIGO.

This may be due to **alcohol, tobacco, carbon monoxide**, quinine, sodium salicylate, or cannabis. The pathogenesis seems to be complex—angiospastic conditions in the brain, congestion of the internal ear, transient cerebral anemia, etc.

The influence of *renal disease* has been clearly shown by Bonnier, who called attention to the frequent occurrence of vertigo in renal cases and proposed as a suitable term in this connection the word *otobrightism*. Here is another circumstance showing how important it is to test the blood-pressure and examine the urine (and the renal functions more generally) in all patients, and in cases of vertigo in particular. The practitioner should keep in mind, at least, that vertigo may be dependent upon high blood-pressure, arteriosclerosis, and uremia.

## V.—REFLEX VERTIGO.

Perhaps it is best to place under this heading the so-called "gastric vertigo" or "**vertigo a stomacho laeso**" of older authors. These cases have been rather overlooked of late One should, however, bear in mind—in general practice—that vertigo, whatever be its precise mode of production, is with exceeding frequency, if not constantly, associated with nausea and vomiting; that it is often manifested in "gastric upsets" of varying cause and origin, and that it frequently yields in these cases to treat-

ment directed toward the alimentary tract, *viz.*, diet, purgation, and stomach washing

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# VOMITING. [*From* vomere, *to vomit.*]

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Vomiting consists of the evacuation of the stomach contents by way of the esophagus and mouth. It constitutes a "reversed deglutition" during which, as in deglutition, the openings of the larynx and nasal cavities into the pharynx are closed As a matter of fact, esophageal vomiting is also a possibility. In contrast with the preceding variety, it is not generally accompanied by nausea and always results in the ejection of food, though sometimes also of varying amounts of blood

True (gastric) vomiting is a reflex of which:

The *center* is in the medulla, in the vicinity of the respiratory center.

The *afferent, sensory nerve-paths* arise mainly:

From the pneumogastric (abdominal stimuli).

From the glossopharyngeal (pharyngeal stimuli).

From the trigeminal (nasal stimuli).

From the cerebral cortex (cerebral stimuli, inflammatory, toxic, or psychic).

The *efferent, motor nerve-paths* follow chiefly:

The phrenic nerve (diaphragm).

The pneumogastric nerve (stomach).

The spinal nerves (muscles of the abdominal parietes, recti, etc.).

The above anatomic and physiologic features account for the fact that vomiting may be either of central origin (meningitis, apomorphine, or revolting impressions) or of peripheral origin (appendicitis, indigestion, pregnancy, or tickling of the uvula)

The vomited material may be either·

1 *Alimentary.* It is extremely important to note the degree of previous digestion of the food vomited, its more or less acid or alcoholic odor, its nature, and especially the greater or less interval between its ingestion and evacuation The mere fact of the presence of food ingested on the preceding day, and especially on the

second preceding day, is a serious indication of stasis with marked probability of pyloric stenosis.

2. *Bilious.*
3. *Mucous,* as in the morning vomiting of alcoholics.
4. *Bloody* (see *Hematemesis*).

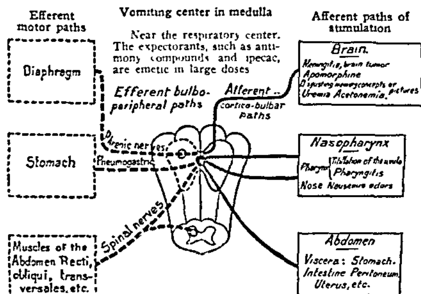



Fig 892—Pathogenesis of vomiting.

5. *Fecaloid,* detected at once by the odor, or if necessary by the pigment tests. Such vomitus is of grave portent. It is nearly always an evidence of intestinal obstruction or occlusion (peritonitis, strangulated hernia, etc.).

The history is always of great importance:

*In some subjects,* vomiting (ranging from simple regurgitation to true vomiting) is an ordinary, habitual event, which occurs almost without effort and with the greatest ease. This is the case in many children, in many alcoholics, in some hearty eaters, and in many gastric neuroses. It is important to record the symptom, but it is usually devoid of any serious significance.

*In others, on the contrary,* vomiting is difficult, distressing, and unusual. Its significance is much greater; the physician must know

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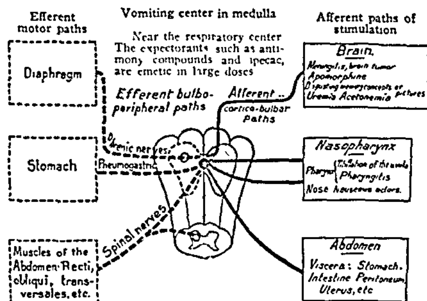



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that he is not to rest content with the superficial and commonplace explanation of indigestion, but is to make a careful examination for the obscure and serious causes of certain cases of vomiting, such as uremia, acetonemia, brain tumor, etc.

In other words, there are some persons in whom quasi-habitual vomiting is almost a negligible event; there are others, however, in whom vomiting is exceptional and of marked clinical significance

In regard to the approximate relative frequency of the commoner causes of vomiting, Cabot gives the following table:

| | |
|---|---|
| Toxemia of pregnancy<br>"Acute dyspepsia" (indigestion).<br>Alcoholism.<br>Seasickness<br>*Onset of infectious diseases*<br>Postoperative shock. | Cases too many and too vaguely enumerable for accurate estimation |
| "Gastritis"<br>Gastric neurosis<br>Acute appendicitis.<br>*Cardiac disease*<br>Peptic ulcer | Intestinal obstruction.<br>Gastric cancer.<br>Uremia<br>*Tabes.* |

*Diseases of the brain* (meningitis, abscess, tumor) are not included; as a matter of fact, the percentage of such cases in the total number of instances of vomiting is very low. The foregoing list, moreover, is by no means complete; it is well, at all events, to mention the vomiting of *meningitis*, of *acetonemia* (precursor of diabetic coma), and of *hepatic* and *renal colic.*

It is not in the scope of this work to review all the possible causes of vomiting and analyze their differential clinical features; it will be sufficient to present a synoptic table of the more common causes with their diagnostic signs.

Some space will, however, be devoted to certain facts chiefly culled from an article by Professor Marfan,[1] relating to a peculiarly difficult task of differential diagnosis, *viz*, that of distinguishing the periodic acetonemic vomiting of children and the vomiting symptomatic of acute appendicitis.

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[1] *Presse méd*, Sept 11, 1916.

**Periodic Vomiting with Acetonemia is not a Form of Appendicitis.**—The phrase "periodic vomiting with acetonemia" is applied to a disorder of childhood characterized by attacks of vomiting, accompanied from the start with pronounced elimination of acetone with the urine and expired air, such attacks generally coming on during apparently satisfactory general health, lasting from a few hours to a few days, and stopping abruptly, being replaced by a state of perfect tolerance of food

They occur almost exclusively in the children of neuroarthritic parents (migrainous, asthmatic, eczematous, lithiasic, obese, gouty, diabetic, and hemorrhoidal). Their occurrence is favored by a diet rich in fats.

Some have asserted that such periodic vomiting with acetonemia is due to an acute attack of appendicitis, constituting an exacerbation of chronic appendicitis The falsity of this view is shown by the fact that the periodic vomiting may occur in children whose appendix has already been removed.

Marfan has long been emphasizing the differential features of these two disorders The following discussion is borrowed from his writings on the subject

"Is the diagnosis between an attack of periodic vomiting and one of appendicitis a matter of great difficulty? In many cases it is not difficult; in a few, it is; occasionally, it is impossible. This is what the writer now wishes to prove by a consideration of the various features on which the diagnosis may be based.

"In distinguishing periodic vomiting from the vomiting caused by acute appendicitis, chief stress is laid on the results of examination of the ileocecal region. One should not, however, neglect any of the other symptoms, although they are of less value.

"In acute appendicitis the temperature is, in general, higher than in the attacks of periodic vomiting, in which it is often normal and in which it is high only under exceptional circumstances and for a short time. In acute appendicitis, acetonemia is inconstant, late in appearing, and often slight, as it is due to the inanition resulting from the vomiting or from the diet ordered by the physician; in periodic vomiting, acetonemia is constant and pronounced, and appears early; it may even be ob-

served before the attack. In acute appendicitis the vomiting and nausea are generally less marked than in the attack of acetonemia. In acute appendicitis, the abdomen is frequently distended; in periodic vomiting, very seldom; on the contrary, in the latter disorder it is usually flat and sometimes scaphoid as in meningitis It should not be forgotten that below the age of five years appendicitis is a comparatively rare affection, being more uncommon in that period of life than periodic vomiting.

"The decisive factors in the diagnosis are, however, supplied by examination of the ileocecal region. If superficial touch elicits cutaneous hyperesthesia of this entire region; if forcible, deep palpation reveals a painful contracture of the muscles of the abdominal wall, and if it induces a well localized or distinctly more pronounced pain at McBurney's point (the middle point of a line joining the umbilicus and the anterior superior spine of the ilium, or slightly outside this point), the disorder present is acute appendicitis. We are not referring to the cases in which the diagnosis is rendered still more obvious by the presence of a swelling in the ileocecal region or by symptoms of diffuse peritonitis Of the foregoing signs, cutaneous hyperesthesia and rigidity are the most important; they are possessed, to my mind, of greater significance even than pain on deep palpation, which is often hard to localize at McBurney's point on account of the resistance offered by the abdominal muscles

"In the attack of periodic vomiting, the abdomen is not painful at any point; it is only after the attack has lasted some days that the muscles of the abdominal wall, having been repeatedly dragged upon by attempts at vomiting, may become the seat, not of actual pain, but of a feeling of soreness, especially at their points of insertion on the ribs

"As a rule, the examination of the abdomen permits of readily making a diagnosis between periodic vomiting and appendicitis. But such is not always the case There are cases in which one is in doubt, and in which one has a right to be in doubt. Following is an example of such a case. A child is seized with vomiting and slight fever, hardly exceeding 38° C.; the ileocecal region is examined; cutaneous hyperesthesia is absent, and rigidity is lacking or is very slight and diffused over the entire abdomen; deep palpation

elicits only trifling pain, without distinct localization, so that the physician questions whether the sensitiveness elicited is not merely due to the pressure exerted by the examining finger; finally, acetonemia appears early and is pronounced In a case of this sort it is very hard to come to any definite diagnostic decision.

"True, sometimes rectal palpation will remove the doubt; if such palpation leads the examiner to feel a tender infiltrated area at the upper portion of the wall of the pelvic cavity on the right side, a decision in favor of the presence of appendicitis should be made. But this sign is frequently lacking, and if it cannot be elicited, the original doubts persist

"There is another class of cases in which the diagnosis remains in suspense; these are the cases in which the results of examination of the ileocecal region change from one attack to the next: In one attack there is nothing wrong in the ileocecal region, acetonemia appears early, and a diagnosis of periodic vomiting is made In the next succeeding attack, there are found abdominal evidences pointing to inflammation of the appendix.

"These cases are readily explained The same subject may be suffering both from periodic vomiting and from appendicitis. An appendicular attack in a predisposed subject may, like any other acute febrile disorder, bring on an attack of periodic vomiting with pronounced acetonemia.

"To recapitulate, in the majority of cases periodic vomiting and acute appendicitis appear in a typical form and are rather easily distinguished Sometimes, however, such differentiation is very difficult, and occasionally it is impossible. The commonest cause of such difficulties consists in the possible coexistence of the two affections in the same subject In the event of doubt, the case should be dealt with as though appendicitis were known to exist, *i e*, if the patient is a child, one should nearly always recommend subsequent removal of the appendix

"In the foregoing presentation, we have referred to cases in which the physician is called upon to settle the question of diagnosis during the attack of vomiting But it may happen that he must make a retrospective diagnosis, *i.e.*, one made a variable time after the occurrence of an attack which he did not witness

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By far the commonest kind of vomica is the pleural vomica, which constitutes a possible termination of *suppurative pleurisy*, interlobar, mediastinal, diaphragmatic, or encysted, sometimes with formation of a *pyopneumothorax*.

The pulmonary vomica, much less common, follows, as the term implies, *abscess of the lung* or *gray hepatization* (suppurative pneumonia); the germ found in the evacuated pus is nearly always the pneumococcus. Some *cavities in the lung* and some *bronchiectatic cavities* may lead to purulent expectoration in such amount as to suggest a vomica.

As an exceptional condition, reference may be made to the *hydatid pulmonary vomica*, or suppurative pulmonary hydatids, which simulate the pleural vomica and can be differentiated from it only by the finding of pieces of hydatid membrane and of echinococcic hooklets in the ejected material

Finally, any variety of *subdiaphragmatic abscess*, whether of hepatic, renal, gastric, or splenic origin, may under exceptional circumstances burrow through the diaphragm and pleura and appear externally in the form of a vomica

In all these disturbances the history of the case and systematic examination will, as a rule, enable the physician very easily to trace back from the symptom, *vomica*, to its pleural, pulmonary, or subdiaphragmatic (hepatic, gastric, renal, or splenic) source.

We believe that it is often very hazardous to make a decision under such circumstances. No doubt there are cases in which examination of the ileocecal region will yield unmistakable evidences; where, the attack of vomiting having terminated a variable time before, deep palpation nevertheless reveals distinct tenderness clearly localized at McBurney's point, and where such palpation also reveals the presence of some degree of muscular contracture strictly confined to the abdominal wall in the right iliac fossa and lacking in the left iliac fossa, one may conclude that there has occurred an attack of acute appendicitis which has left behind it some degree of subacute or chronic inflammation of the appendix But such cases are rather uncommon. *Usually examination of the right iliac fossa after the attack* shows no abnormal condition, or else, examination for tenderness at McBurney's point or for rigidity gives such indefinite and inconclusive results that they cannot be interpreted without risk of error Hence, where the practitioner has not actually seen the attack, he should leave to the physician who may have seen it the responsibility of reaching a definite decision If, however, circumstances are such that a decision must be made even under the conditions already described, the family should be carefully apprised of the fact that the decision made can be based only upon conjecture."

* * *

It seems impracticable to conclude an article on the semeiology of vomiting without a brief additional consideration of the subject of "Vomica." By the term *vomica* is meant the evacuation through the respiratory tract of an accumulation of pus in such amount that it suggests *vomiting* (*vomere*, to vomit).

The condition is entirely different from vomiting, inasmuch as the evacuation of fluid takes place through the respiratory tract. A vomica is, strictly speaking, an expectoration of pus in large amount. But from the standpoint of gross symptomatology the occurrence is a vomiting of pus—a sudden ejection through the mouth of a considerable amount of purulent fluid. generally accompanied by paroxysmal cough and a degree of suffocation sometimes bordering on complete asphyxia.

| | | | | |
|---|---|---|---|---|
| Onset of an infectious disease (pneumonia, smallpox, scarlet fever, etc.). | History of exposure to the disease | *Nothing characteristic.* | Fever. Leucocytosis Loaded urine. Evidences of the infectious disease (pneumonia, measles, scarlet fever, malaria, etc.). | That of the infectious disorder. |
| Cardiac disorders in the stage of decompensation. | Heart disease. | Nothing characteristic. | Evidences of heart weakness or failure; various evidences of circulatory stasis. | Improvement under diet, rest, etc. |
| Gastric ulcer. | That of dyspepsia with hyperchlorhydria<br>Age, 20 to 40 years | Chronic, intermittent, a few hours after meals Often of high acidity Sometimes hematemesis | Ulcer pain Local sensitiveness Pain relieved by vomiting or alkalies Blood in the feces | Improvement under diet, rest, or operation. |
| Cancer of the stomach. | Onset relatively late in life, after the fortieth year | Late *vomiting of mucus* and food, sometimes greatly delayed, the food having been taken 18, 24, or 36 hours before Stasis and sometimes hematemesis Frequently achlorhydria | Dilated stomach Stasis of food Blood in the stools Tumor of the stomach (insufflation, X-rays) | Progressive cachexia, possible improvement by operation |
| Intestinal obstruction and occlusion (strangulated hernia, etc Peritonitis). | Chiefly in elderly persons<br>Constipation, spastic or atonic.<br>Often previous attacks of obstruction. | Food, then bile, then fecal vomiting. | Abnormal peristalsis Meteorism. Stercoral or intestinal tumor. Look for a hernia. Practise rectal palpation. | Improved by deobstructive treatment or operation. |

| CAUSES | HISTORY | KIND OF VOMITING | OTHER EVIDENCES | CLINICAL COURSE |
|---|---|---|---|---|
| **Indigestion. "Acute Dyspepsia."** | Recent meal of excessive amount or unusual composition | Food recently ingested Bile | | Rapid recovery under diet and rest |
| **Alcoholism** acute. | Recent ingestion of alcoholic beverages | Odor of alcohol, mucus, and food | | Rapid recovery under diet and rest |
| **Alcoholism** chronic (chronic gastritis) | Habitual abuse of liquor | Morning vomiting of mucoid material | The usual stigmata of chronic alcoholism (tremor, liver disturbances, chronic dyspepsia, etc ) | Rapid improvement upon abstinence from liquor |
| **Acute appendicitis.** | Sometimes dyspepsia, former attacks | Nothing characteristic | Characteristic pain in the right iliac fossa **(see this sign)** Leucocytosis. | Absorption or operation or complications |
| **Gastric neuroses.** | Neuropathic history | Generally food, or quite abnormal or stereotyped | Absence of gastric symptoms Presence of neuropathic indications | Cure by suggestion and reëducation. |
| **Hepatic colic, renal colic, etc** | History of hepatic or nephritic disturbance | Nothing characteristic. | Sudden onset Pain of special, characteristic situation (see *Pain*) | That of the colic |
| **Pregnancy (always to be thought of in women).** | That of pregnancy. | Especially in the morning, mucoid material, food, sometimes periodic, sometimes incoercible. | Enlargement of the uterus Cessation of menstruation, etc | Generally ceases after the earlier months, sometimes incoercible. |

# INDEX OF THE PRINCIPAL CLINICAL SIGNS.[1]

## ARRANGED ACCORDING TO THE NAMES OF THE AUTHORS CONCERNED.

### A

**Abderhalden Reaction.** *Biochemistry*

A reaction having for its purpose to demonstrate the proteolytic ferments set free in the blood upon introduction into the system of incompletely elaborated organic products. Has been used for the diagnosis of pregnancy. Technic difficult. Results questionable.

**Abrams's Reflexes.** *Physiology*

Cardiopulmonary reflexes awakened by peripheral stimulation (skin, muscles, and mucous membranes). Percussion over the precordium causes a reduction of the area of heart dullness through dilatation of the lungs and contraction of the heart.

**Adams-Stokes Disease.** *Syndrome*

Featured by a marked slowing of the pulse rate (to 30 or 40) with dizziness, epileptoid seizures, and some- [illegible]

**Addison's Disease.** *Endocrinology*

Characterized by a variable degree of asthenia, brown pigmentation of the skin and mucous membranes, and low blood-pressure. It is due to disease of the adrenal glands.

**Aran-Duchenne Type.** *Neurology.*

Progressive muscular atrophy beginning with the muscles of the hands.

**Argyll-Robertson Pupil.** *Neurology*

An early sign of tabes dorsalis, consisting of disappearance of the pupillary light reflex with conservation of the accommodation reflex.

**Arthus's Phenomenon.** *Serology.*

Local skin disturbances (eruptions, erythema, edema) following repeated injections of foreign serums.

**Aschner's Sign.** *Neurology.*

The oculocardiac reflex, consisting of a slowing of the pulse rate by 5 to 15 or 20 beats per minute upon compression of the eyeball. The reflex is enhanced in vagotonic states. Acceleration of the pulse rate (inversion of the reflex) occurs in sympatheticotonic states.

**Avellis's Syndrome.** *Neurology*

Hemiplegia of the soft palate and paralysis of the recurrent nerve on the same side.

### B

**Babinski's Sign.** *Neurology.*

This sign of disease of the spinal cord (pyramidal tract) consists in extension of the big toe when the sole of the foot is mechanically stimulated by drawing the point of a pin over it.

**Baccelli's Sign.** *Lungs*

Whispering pectoriloquy. Distinct transmission of the whispered voice to the ausculting ear on the side of a pleural effusion.

**Baccelli's Anguloscapular Sign.** *Lungs.*

Reduced motion of the scapula during deep inspiration in tuberculosis of the apex.

**Balfour's Disease.**

Osseous sarcomatosis affecting chiefly the bones of the cranium and face and attended with greenish discoloration (chloroma).

[1] Prepared with the assistance of Dr. Prevel.

| CAUSES | HISTORY | KIND OF VOMITING | OTHER EVIDENCES | CLINICAL COURSE |
| --- | --- | --- | --- | --- |
| **Uremia (arterio-sclerosis).** | That of uremia. | *Nothing characteristic* | *Those of uremia.* Albuminuria, high blood-pressure, azotemia, headache, vertigo, *heart disturbances, hemorrhages,* etc. | Improved by starvation, venesection, and purgation. |
| **Tabes dorsalis.** | Syphilis 6 to 20 years before. | Sudden *onset* *Abrupt* cessation after paroxysmal attacks of a few days' duration. | *Accompanied by sharp pains of* the ulcer type Usual signs of tabes Reflex disturbances, *astasia, ataxia, urinary* disturbances, etc, lightning pains | Relieved with difficulty by morphine. |
| **Diabetes (with imminent coma and *acetonemia*).** | Diabetes | Sometimes odor of acetone, chloroform, or apples | Dyspnea Increasing stupor Presence of sugar, acetone, and diacetic acid in the urine. | Usually fatal, sometimes improved by *alkalies.* |
| **Meningitis.** | Headache<br>Sore throat<br>Emaciation<br>Tuberculous infection. | Recurring without effort and without relation to ingestion of food | Headache<br>Kernig's sign.<br>Fever.<br>(Lumbar puncture.) | *Usually fatal.* |
| **Migraine.** | History of migraine<br>*More or less periodic* recurrence of the attacks. | Food and bile | Headache | Allayed by analgesic drugs *and tending to* pass off with advancing age. |

bacteriolysis, etc.) with the corresponding antigen

When heated to 55° C the specific serum loses its specific reacting power. Brought in contact with the antigen, *it is inactivated.*

It is reactivated by the addition of normal serum, which supplies to it what it lacks, *viz., complement.* Brought in contact with the antigen, it now again yields the specific reaction.

The complement of the normal serum thus utilized is permanently fixed in the reaction, *i.e., it is deviated* and cannot serve again for further reactivation.

**Bouchard's Nodes.** *Metabolism.*

Thickening of the second joints of the fingers in certain subjects with dilatation of the stomach.

**Boudin's Law.** *Tuberculosis*

Antagonism of malaria and tuberculosis, possibly dependent upon hypertrophy of the liver.

**Bouillaud's Laws.** *Rheumatism*

Endocarditis and joint inflammation are usually concomitant in acute articular rheumatism with severe and multiple manifestations

They are not concomitant in cases with only partial or mild manifestations.

**Bouveret's Disease.** *Cardiology*

Marked tachycardia (180, 200, or higher) accompanied by low blood-pressure and elevation of temperature, coming on in sudden attacks of variable duration, from a few minutes to several days (heart failure). Paroxysmal tachycardia

**Bozzolo's Sign.** *Cardiology*

Visible pulsations of the nostrils in some cases of thoracic aneurysm

**Brandt's Method.** *Therapeutics*

Treatment of typhoid fever by the systematic use of cold baths

**Bright's Disease.** *Kidneys*

Chronic nephritis with albuminuria, high blood-pressure, and terminal edema

**Bright's Sign.** *Peritonitis.*

Peritoneal friction sounds.

**Briquet's Gangrene.** *Lungs.*

Gangrene of the bronchi in the course of bronchiectasis.

**Brissaud and Sicard, Syndrome of.** *Neurology.*

Motor disturbances on one side of the body with facial hemispasm on the opposite side.

**Broadbent's Sign.** *Cardiology.*

Systolic retraction of the left posterior aspect of the chest at the level of the diaphragm; a sign of pericardial adhesion

**Broca's Aphasia.** *Neurology.*

Motor aphasia resulting from disease of the lower portion of the left third frontal convolution

**Brown-Séquard's Method.** *Endocrinology.*

Opotherapy The administration of extracts of organs in disease of the identical organs to make up for their deficiencies or to stimulate them A procedure based on the fact that the vascular glands produce an internal secretion

**Brown-Séquard's Syndrome.** *Neurology.*

Unilateral disease of the spinal cord causing hemiparaplegia with hemianesthesia on the opposite side.

**Brudzinski's Signs.** *Neurology.*

Signs of meningitis A reflex movement of flexion or extension of the lower extremity is obtained by strongly flexing the limb of the opposite side (contralateral reflex).

Flexion of the lower extremities is obtained upon flexing the neck.

**Bryson's Sign.** *Graves's disease.*

Deficient chest expansion during inspiration in cases of Graves's disease.

## C

**Charcot's Disease** *Neurology.*

Spastic paralysis in conjunction with progressive muscular atrophy

**Charcot-Marie's Sign.** *Neurology*

The rapid tremor of exophthalmic goiter.

**Bamberger's Sign.** *Neurology.*
A disturbance of sensation in tabetics Stimulation of a restricted area of skin is referred by the patient to the opposite side of the body (allochiria)

**Banti's Disease.** *Liver.*
*Hepatic cirrhosis* with ascites, progressive anemia, and enlargement of the spleen.

**Barany's Sign.** *Otology*
Disappearance of hydro-caloric vestibular nystagmus in severe disease of the internal ear

**Bard and Pic's Syndrome.** *Pancreas.*
In cancer of the head of the pancreas the following conditions are present in combination Chronic, progressive jaundice, cachectic emaciation, and dilatation of the gall-bladder.

**Bard's Sign.** *Neurology*
Serves to distinguish organic from congenital nystagmus In the first instance, the oscillations of the eye increase as the patient follows the physician's finger moved before his eye from right to left and then from left to right In the second instance, the oscillations disappear

**Barlow's Disease.** *Pediatrics.*
A disease of early childhood characterized by anemia with bone pains (subperiosteal hemorrhages)

**Basedow's Disease.** *Endocrinology.*
Thyroid enlargement with tachycardia, exophthalmos, and tremor

**Baumès, Law of.** *Syphilography*
A syphilitic father may procreate a syphilitic child without the appearance of specific manifestations in the mother and without risk to her in nursing her infant.

**Beard's Disease.** *Neurology*
Neurasthenia. A neuropathic symptom-complex consisting of a variable degree of insufficiency of the muscular, circulatory, secretory, and digestive functions, with insomnia and general asthenia. Met with in over-*worked individuals.*

**Bell's Sign.** *Neurology.*
Displacement of the eyeball upward and outward when the patient attempts to lower the *paralyzed* upper eyelid (peripheral facial paralysis).

**Bence-Jones's Disease.** *Sarcoma*
Concomitant albumosuria and sarcoma.

**Benedikt's Syndrome.** *Neurology*
Hemiplegia with paralysis of the oculomotor nerve of the opposite side (disease of the cerebral peduncle).

**Bergeron's Disease.** *Pediatrics.*
Infantile chorea with rhythmic movements in more or less rapid repetition.

**Besredka's Method.** *Vaccine therapy.*
Antianaphylactic vaccination carried out by administering an injection of 0 1 cubic centimeter of serum two hours before the therapeutic injection

**Bier's Method.** *Therapeutics*
Use, in the treatment of inflammatory conditions, of venous hyperemia induced by elastic constriction of the veins (in the extremities) or by application of suction cups of varying shape and size.

**Biett's Ring.** *Dermatology*
A small ring of white epidermis frequently present about the skin lesions of secondary syphilis

**Bird's Disease** *Metabolism*
The sum of the digestive, urinary, circulatory, and nervous disturbances resulting from the oxalic diathesis

**Bockhart's Impetigo.** *Dermatology*
Vesiculopustules always developing about a hair follicle. A skin disorder of childhood

**Bonfils's Disease.** *Blood.*
Proliferation of the lymphoid tissue without increase in the white blood cells (aleukemic or simple *lymphadenia*).

**Bordet and Gengou Reaction.** *Serology.*
A complete specific serum (*containing* antibodies following injection of an antigen bacteria, cells, or toxins) yields a specific reaction (hemolysis,

**Finsen's Method.** *Therapeutics*
Treatment of skin disorders by means of selected light rays

**Fochier's Method.** *Therapeutics*
Treatment by means of artificial aseptic abscesses (fixation abscesses)

**Friedreich's Disease.** *Neurology.*
Hereditary locomotor ataxia appearing in childhood and persisting indefinitely.

**Fröhlich's Syndrome.** *Endocrinology*
Obesity associated with an infantile condition of the sexual organs, due to pituitary insufficiency

**Fürbringer's Sign.** *Subphrenic abscess*
Serves to differentiate subdiaphragmatic abscess from abscess in the chest. A needle passed into the abscess cavity is displaced with the respiratory movements in the former case and not in the latter

## G

**Gaucher's Disease.** *Spleen*
Primary epithelioma of the spleen

**Gerhardt's Test.** *Urine*
Portwine color of the urine upon addition of ferric chloride, pointing to the presence of diacetic acid

**Gmelin's Test.** *Urine*
An emerald green ring formed at the surface of contact between urine and nitric acid without application of heat; points to the presence of bile pigments in the urine

**Godelier's Law.** *Tuberculosis*
Tuberculization of the pleura always occurs when tuberculosis of the peritoneum exists

**Gordon's Sign.** *Neurology.*
Extension of the great toe upon compression of the muscles of the calf; points to disease of the pyramidal tract.

**Gradenigo's Syndrome.** *Otology*
Paralysis of the sixth cranial nerve (abducens) during acute otitis media

**Graefe's Sign.** *Endocrinology.*
Dissociation of the movements of the upper lid and eyeball when the eye glances downward A sign of exophthalmic goiter

**Grancher's Disease.** *Lungs.*
Massive congestion of the lung without pleural effusion, but yielding clinical signs similar to those of pleurisy

**Graves's Disease** *Goiter.*
Exophthalmic goiter or Basedow's disease

**Graves's Sign.** *Gout.*
Abnormal sensitiveness of the dental nerves in gouty individuals, causing them to grind the teeth

**Grocco's Triangle.** *Percussion.*
A triangular area of paravertebral dullness at the base of the thorax on the side opposite that of pleurisy. Ascribed to displacement of the mediastinal structures

**Guérin's Law** *Rachitis*
Rachitic deformities begin in the lower portions of the body

## H

**Hahnemann's Method.** *Therapeutics.*
Homeopathy

**Hanot's Disease.** *Liver.*
Hypertrophic cirrhosis with chronic jaundice

**Harley's Disease.** *Blood.*
Paroxysmal hemoglobinuria coming on on account of exposure to cold

**Harrison's Groove** *Rickets.*
Observed in rachitic subjects during deep inspiration, between the chest and the upper portion of the abdomen, at the level of insertion of the diaphragm.

**Head's Zones.** *Neurology.*
Innervation of the visceral structures and skin surface in corresponding zones Cutaneous hyperesthesia in definite zones points to disease of the corresponding deep-seated organs

**Cheyne-Stokes Breathing.** *Lungs.*
A type of breathing comprising a series of respirations of increasing amplitude, then of decreasing amplitude, followed by a varying period of apnea and resumption of the increasing respirations

**Chvostek, Jr.'s Sign.** *Tetany*
Increased mechanical irritability of the motor nerves in tetany

**Clapton's Sign.** *Copper poisoning.*
A greenish line on the gums in copper poisoning

**Claude-Bernard-Horner, Syndrome of** *Neurology*
A sympathetic ocular syndrome characterized by enophthalmos, myosis, and vasomotor disturbances on the same side of the face (elevation of local temperature and sweating)

**Colrat's Test.** *Liver*
Alimentary glycosuria, demonstrated by having the patient take 150 grams of glucose on an empty stomach and tracing the sugar in the urine on the same day (an indication of insufficiency of the liver)

**Corrigan's Disease.** *Cardiology.*
Aortic insufficiency, of syphilitic or rheumatic origin

**Corrigan Pulse.** *Cardiology*
A bounding and brief pulse Abrupt ascent, rapid turn and quick descent of the pulse tracing Aortic insufficiency due to endocarditis

**Courvoisier and Terrier, Law of.** *Liver*
Atrophy of the gall-bladder in the presence of obstruction of the common bile-duct by a stone; dilatation of the gall-bladder in other kinds of obstruction

**Cruveilhier's Disease.** *Stomach.*
Gastric ulcer.

D

**Damoiseau's Curve.** *Pleura.*
A curve with its convexity uppermost, formed by the surface of pleural effusions.

**Déhio's Test.** *Cardiology.*
Testing for cardiac acceleration in bradycardia upon injection of 1 milligram of atropine sulphate If the test is positive, the bradycardia is of nervous origin, if negative, of cardiac origin.

**Dejerine-Klumpke, Syndrome of.** *Neurology*
Paralysis of the lower nerve-roots of the brachial plexus, accompanied by myosis and enophthalmos.

**Dubini's Chorea.** *Neurology*
Chorea marked by convulsive attacks which are followed by paralysis and coma

**Duchenne's Disease.** *Neurology*
Tabes dorsalis

**Duguet's Sign.** *Cardiology*
Ulcers on the pillars of the soft palate in typhoid fever

**Duroziez's Disease.** *Cardiology.*
Uncomplicated mitral stenosis

**Duroziez's Sign.** *Cardiology.*
A double murmur heard with a stethoscope exerting gentle pressure over the femoral artery.
A sign of aortic regurgitation.

E

**D'Espine's Sign.** *Auscultation.*
Bronchophony of the whispered voice elicited by auscultation over the spinal column between the scapulæ (a sign of intertracheobronchial glandular enlargement)

**Erb's Sign.** *Neurology*
Enhanced electric excitability of the muscles and nerves in tetany.
Disappearance of the pupillary response to pain in tabes

F

**Fallot's Disease.** *Cardiology.*

**Lane's Disease.**
Chronic intestinal stasis.

**Lasègue's Sign.** *Neurology*
A sharp pain elicited in the buttock by flexion of the thigh on the pelvis with the lower extremity extended An indication of sciatica.

**Leyden-Mobius Type.** *Myology*
Muscular atrophy beginning in the lower limbs and later gradually involving the upper extremities

**Litten's Sign.** *Lungs*
Reduced mobility of the diaphragm on the affected side in pulmonary tuberculosis

**Little's Disease.** *Neurology*
Congenital spastic paraplegia occurring in premature infants or following unusually difficult labor

**Ludwig's Angina.** *Mouth*
Infectious cellulitis of the floor of the mouth.

## M

**Madelung's Disease.**
Hand in the valgus position, with prominence of the head of the ulna and palmar subluxation of the hand

**Marey's Law.** *Cardiology*
Tachycardia generally accompanies low blood-pressure and bradycardia high blood-pressure

**Marie's Disease.** *Dystrophy*
Enlargement of the hands, feet, and face.

**Martinet's Laws.** *Circulation*
Dividing the daily output of urine by the mean differential (pulse) pressure gives a result equal to or exceeding one-fourth liter in the healthy subject on a normal diet.
Persistence of the result below 0 200 liter is characteristic of renal sclerosis

**Martinet's Syndrome.** *Circulation*
Hyposphyxia, characterized by low blood-pressure and an absolutely or relatively high blood viscosity, with small pulse and slowed circulation.

**Menetrier's Syndrome.** *Thoracic duct*
Signs of pressure upon the thoracic duct: Firm edema of the lower portion of the body, the chest and the *left* arm, with peritoneal and pleural effusions on the left side

**Ménière's Syndrome.** *Otology.*
Vertigo, with various sounds heard by the patient Reduction of auditory acuity An indication of internal ear disease

**Mikulicz's Disease.** *Glands*
Enlargement of the lacrymal and salivary glands on both sides, with suppression of their secretion but without local pain, believed frequently due to leukemia.

**Millard-Gubler Syndrome.** *Neurology*
Hemiplegia on one side with facial paralysis on the other.

**Möbius's Disease.** *Neurology.*
Ophthalmoplegic migraine.

**Mobius's Sign.** *Endocrinology.*
Difficulty of convergence of the eyes in exophthalmic goiter

**Morton's Disease.** *Feet*
Metatarsalgia frequently following fatigue

**Morvan's Disease.** *Leprosy.*
Felon accompanied by anesthesia of the fingers, considered of leprous nature

**Murphy's Method.** *Therapeutics.*
Rectal administration, drop by drop, of glucose or saline solution in high fever or after surgical operations

**Musset's Sign.** *Cardiology.*
Rhythmic jerking movements of the head, synchronous with the heart beats, in patients with aortic regurgitation. [Alfred de Musset is said to have exemplified this sign]

## N

**Negri Bodies.** *Rabies.*
Found in the central nerve cells of animals that have succumbed to rabies. Considered specifically related to the disease.

**Heberden's Nodes** *Rheumatism.*
Nodes about the terminal phalangeal joint in chronic rheumatism.

**Heine-Kreysig's Sign.** *Pericardial adhesion.*
Systolic depression of the intercostal spaces indicating pericardial adhesion (cardiac symphysis).

**Heine-Medin's Disease.** *Infections*
An infectious, epidemic disorder resembling infantile paralysis.

**Heine-Sanders' Sign.** *Pericardial adhesion.*
Wave-like motion of the chest-wall extending beyond the boundaries of cardiac dullness and most marked in the epigastric region Characteristic of pericardial adhesion

**Hérelle's Phenomenon.** *Bacteriology.*
Given a 24-hour culture of Shiga dysentery bacillus in alkaline peptone bouillon and a filtrate (from a Chamberland filter) of stools of a person convalescent from dysentery, a few drops of this filtrate *will clear the turbid suspension of* bacilli in a few hours (6 to 18 hours at 37° C) The culture thus cleared no longer contains dysentery bacilli Bacteriolysis has taken place

**Herxheimer's Reaction.** *Syphilis*
Temporary accentuation of syphilitic manifestations as a result of mercurial or arsenical treatment.

**Hirschsprung's Disease.** *Colon.*
Congenital megacolon accompanied by constipation and abdominal enlargement in young children.

**Hodgson's Disease.** *Cardiology*
Aortic insufficiency of arterial origin

**Hutchinson Teeth.** *Teeth.*
Dental deformity characterized by semicircular notches in the free margins of the median upper incisors with narrowing of the necks of the teeth A sign of congenital syphilis.

## J

**Jaccoud's Sign.** *Cardiology*
A sign of pericardial adhesion *consisting of a rolling movement* in the precordial region.

**Jellineck's Sign.** *Dermatology.*
Discoloration of the eyelids in Basedow or nervous subjects

## K

**Karell's Treatment.** *Therapeutics*
Marked reduction in the intake of solid and liquid food (800 grams of milk per diem); used in certain cases of heart weakness.

**Kernig's Sign.** *Neurology.*
Flexion of the legs on the thighs when the lower limbs are placed at a right angle with the trunk (sitting posture in bed). A sign of spinal meningitis.

**Kienböck's Sign.** *Pleura*
*Fluoroscopy showing* a rise of the diaphragm at the time of inspiration on the side of an effusion of fluid and air in the pleura.

**Klippel's Disease.** *Neurology.*
A species of rapidly progressive *general paralysis occurring* in old men (dementia, stroke, and paralytic phenomena).

**Klippel-Feil's Syndrome.**
Reduced number of segments of the spinal column (neckless men).

**Kussmaul Breathing.** *Respiration.*
A kind of breathing characterized by a prolonged inspiration followed by a pause and a brief expiration, followed by a second pause. Met with in diabetic coma

## L

**Laennec's Cirrhosis.** *Liver.*
Atrophic cirrhosis

**Landouzy-Dejerine Type.** *Myology.*
A form of progressive muscular atrophy of childhood beginning with the face, shoulders, and arms.

**Landry's Disease.** *Neurology*
Paralysis of the lower extremities of an acute type, soon involving the trunk and causing death *within a few days.*

**Lane's Disease.**
Chronic intestinal stasis.

**Lasègue's Sign.** *Neurology*
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**Negro's Sign.** *Neurology.*
A sign of peripheral facial paralysis: Elevation of the eyeball is more marked on the paralyzed side when the patient looks upward with the head motionless.

## O

**Oertel's Method.** *Therapeutics.*
Treatment by graded exercise (walking on level ground or inclines) in chronic heart disorders.

**Oliver's Sign.** *Aortic aneurysm*
Movements of the larynx from below upward, synchronous with cardiac systole, in subjects suffering from aneurysm of the arch of the aorta.

**Oppenheim's Sign.** *Neurology.*
A sign of disease of the pyramidal tracts Ascent of the great toe when pression is exerted from above downward over the muscles of the anteroexternal aspect of the leg

## P

**Paget's Disease of the Bones.** *Bones*
Marked thickening of the bones of the skull and extremities Believed an indication of inherited syphilis.

**Paget's Disease of the Nipple.** *Breast*
A malignant tumor starting at the nipple, in old women

**Parkinson's Disease.** *Neurology*
A disease characterized by rigid posture of the body, a facies as of surprise, and a peculiar (pill-rolling) tremor of the fingers.

**Parrot's Disease.** *Bones*
Syphilitic epiphyseal detachment.

**Parrot's Law.** *Tuberculosis*
A tuberculous lesion of the bronchial lymph-nodes is always accompanied by a pulmonary lesion of the same nature.

**Parrot's Sign.** *Meningitis*
Dilatation of the pupil when the skin is pinched in meningitis

**Pavy's Disease.** *Albuminuria.*
Intermittent cyclic albuminuria in young subjects, occurring in the daytime.

**Perret and Devic, Signs of.** *Pleura.*
Signs of pleurisy at the base of [illegible]

[illegible]

**Pettenkoffer's Test.** *Urine.*
Purple violet color of the urine when treated with sulphuric acid in the presence of sugar, points to the presence of bile acids

**Pfuhl's Sign.** *Pleura.*
Shows whether an effusion is above or below the diaphragm In the first instance the pressure in the manometer connected with the trocar rises during *inspiration; in* the second instance, it falls.

**Porgès's Reaction.** *Serology.*
Precipitation of serum in the presence of a solution of sodium glycocholate Claimed to indicate syphilis

**Pott's Disease.** *Bones.*
Tuberculosis of the vertebræ.

**Profeta, Law of.** *Syphilis*
A syphilitic mother may nurse her healthy infant without risk of infecting it.

## Q

**Quincke's Disease.** *Dermatology.*
Hereditary acute paroxysmal edema, unattended with constitutional disturbance

## R

**Raynaud's Disease** *Circulation*
Disturbances of the circulation in the extremities (cyanosis, local asphyxia, "dead finger"), which may lead to dry gangrene.

**Recklinghausen's Disease.** *Dermatology.*
Cutaneous and nervous tumors (neurofibromata) accompanied by pigmentation of the skin.

**Reclus's Disease.** *Breast.*
Presence of many small shot-like cysts in the breast.

**Revilliod's Phenomenon.** *Hemiplegia*
Inability, in organic hemiplegia, to close the eye on the paralyzed side without at the same time closing that on the well side

**Rinné's Test.** *Otology*
Rinné positive when the sound of the tuning-fork is heard better by air conduction than by mastoid (bone) conduction. Rinne negative when the sound is better conducted by bone than by air

**Rivalta's Test.** *Serology*
A few drops of effused fluid cause a turbidity when dropped in water acidulated with acetic acid if the effusion is of inflammatory nature

**Roger's Disease.** *Cardiology*
Congenital communication between the ventricles of the heart, unattended with dyspnea nor cyanosis when the subject is at rest

**Romberg's Sign.** *Neurology*
An indication of tabes dorsalis: Loss of equilibrium when, with the eyes closed, the heels are brought together.

**Rosenbach's Sign.** *Neurology.*
Persistence of the abdominal reflex in hysteric hemiplegia, in spite of the anesthesia of the skin

**Rosenheim's Sign.** *Perigastritis*
Friction sounds heard on auscultation over the left hypochondrium in cases of fibrous perigastritis

**Ruault's Sign.** *Respiration*
Diminished amplitude of respiration on the affected side in incipient pulmonary tuberculosis.

## S

**Sahli's Test.** *Pancreas*
Where the pancreas is [illegible]

**Sicard's Method.** *Neurology.*
Treatment of certain disorders by the introduction of solutions of drugs into the epidural space.

**Sieur's Sign.** *Pleurisy.*
A ringing, metallic sound elicited in pleural effusion by percussing the opposite point with two coins, the one placed against the thorax and the other used as pleximeter.

**Souques's Sign.** *Neurology.*
Spreading of the fingers when a patient with organic hemiplegia attempts to raise the paralyzed arm (incomplete flaccid hemiplegia).

**Stellwag's Sign.** *Endocrinology.*
Incomplete closure of the eyes in exophthalmic goiter

**Stokes-Chopart, Law of.** *Inflammation.*
The muscles underlying an inflamed mucous membrane or serous surface are in a paralyzed state.

**Straus's Sign.** *Neurology.*
In severe peripheral facial paralysis the sweating induced by pilocarpine is delayed.

## T

**Thomsen's Disease.** *Myology.*
Spastic contraction of the muscles when voluntary movements are attempted

**Thure-Brandt Posture.** *Abdomen.*
Posture in which the abdominal wall is relaxed Dorsal decubitus, lower extremities flexed, and buttocks raised, the patient taking deep inspirations

**Traube's Law.** *Circulation.*
Interstitial nephritis always tends to bring about hypertrophy of the left ventricle

**Troisier's Ganglion.** *Lymphatics*
Glandular enlargement above the clavicle on the left side in cancer of the stomach

**Trousseau's Sign.** *Neurology.*
Contracture obtained by exerting pressure on nerves or vessels in tetany.

## V

**Valleix's Laws.** *Neurology*

Governing the location of pain in neuralgia: The painful areas are to be found at the points of emergence and in regions where the nerve ramifications become superficial.

**Valsalva Test.** *Otology.*

By attempting to blow out air with the mouth and nose closed the patient inflates the tympanic cavity.

**Vaquez's Disease.** *Blood*

A disorder characterized by a marked increase in the number of red cells, together with cyanosis and splenic enlargement.

**Vincent's Angina.** *Tonsils*

Subacute tonsillitis with a diphtheroid exudate and associated with the presence of fusiform bacilli

**Volkmann's Contracture.** *Neurology*

Independent retraction of a muscle Ischemic paralysis.

**Vulpian's Law** *Neurology*

In hemiplegia the patient turns his head and eyes toward the side of the lesion, which he appears to look at

## W

**Wassermann Reaction.** *Serology.*

The Bordet and Gengou reaction of fixation applied in the diagnosis of syphilis

**Weber's Test.** *Blood*

A test for traces of blood in the feces or in vomitus Acetic acid, ether, fresh tincture of guaiac, and hydrogen peroxide solution yield a blue color.

**Weber's Test.** *Otology*

A test of the hearing carried out by applying a tuning-fork over the forehead If conduction is better [illegible]

[illegible]

**Werlhoff's Disease.** *Blood*

Cryptogenic purpura and hemorrhages, unattended with fever and prognostically favorable.

**Wernicke's Sign.** *Neurology*

Consists in that, in a subject with bilateral homonymous hemianopsia, pupillary response when a beam of light strikes the blind half of the retina occurs only when the lesion involves the optic fibers beyond the thalamus. In the opposite event, a response is obtained only by stimulating the normal half of the retina

**Westphal's Sign.** *Neurology.*

An early sign of tabes dorsalis: Loss of the patellar reflex.

**Woillez's Disease.** *Lungs*

Acute pulmonary congestion giving rise to symptoms similar to those of pneumonia.

# INDEX TO VOLUMES I AND II.

